ADMISSION TEST AS A SCREENING TEST TO PREDICT FETAL OUTCOME

DISSERTATION SUBMITTED FOR

M.D (OBSTETRICS AND GYNAECOLOGY)

FEBRUARY – 2006

MADURAI MEDICAL COLLEGE

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CERTIFICATE

This is to certify that, this dissertation titled **"ADMISSION TEST AS A SCREENING TEST TO PREDICT FETAL OUTCOME"** submitted by **Dr. Helen. P** for M.D.(Obstetrics and Gynaecology) is a bonafide research work carried out by her under our direct supervision and guidance.

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CONTENTS

Sl.No	Title	Page .No
1.	INTRODUCTION	1
2.	AIM OF STUDY	3
3.	REVIEW OF LITERATURE	4
4.	FETAL PHYSIOLOGY – CONTROL OF FETAL HEART RATE	10
5.	ELECTRONIC FETAL HEART MONITORING. BASICS AND CLINICAL SCENARIOS	14
6.	ADMISSION TEST	36
7.	MATERIALS AND METHODS	41
8.	RESULTS AND ANALYSIS	44
9.	DISCUSSION	66
10.	SUMMARY	72
11.	CONCLUSION	74
	BIBLIOGRAPHY	
	PROFORMA	
	MASTER CHART	

1. INTRODUCTION

The goal of antepartum fetal surveillance is to prevent fetal death. Each and every fetus has a potential risk of intrapartum hypoxia or birth injury and an optimal outcome can be concluded only at the end of labour. However any definite insult due to the process of labour can only be identified on long-term follow up. Of the small number of babies with neurological problems after birth only few can be attributed to the event of labour, although only a few are affected at birth.

A live healthy baby is the prime goal of the parents, the obstetrician, and the state.

In promoting safe motherhood as defined by the World Health Organization, our objectives must be to optimize the

(i) Health of the mother

(ii) Health of the off spring

(iii) Emotional satisfaction of the mother and her family.

Assessment on admission helps us to look carefully for high risk factors particularly undetected & new factors that have appeared.

Two problems have to be solved during assessment. Even after vigorous selection based on a known Antenatal risk classification system fetal morbidity and mortality tends to occur in the so – called Low Risk Groups (Hobel et al

1973). This leaves us with the task of determining who is at low risk. ***A new system must be developed to identify those who are at risk in labour by means of a test "Admission test".***

Next problem we face is the difficulty in providing one to one care to offer optimal standards of intermittent auscultation with inadequately trained man power. For good result with auscultation one has to listen to the fetal heart rate for one minute every 15 minutes perfectly after a contraction in the first stage of labour and after every 5 minutes in the 2nd stage of labour. This may not be feasible in many centres.

Routine electronic fetal heart rate monitoring in labour has become an established practice in the labour ward. In labour ward with few monitors selection of patients for continuous monitoring is necessary. As intrapartum fetal morbidity and mortality are also common in a low risk population, this problem can be over come to some extent by an Admission test.

Admission test is a good screening test because it is a simple test that can be done by midwives, can be rapidly done with in 20 -40 minutes, having high acceptability by the pregnant mothers, can be repeated at any time and high validity and so it can be used as a good intra partum screening test.

2. AIM OF THE STUDY

1. Evaluation of admission test in high risk and low risk groups.
2. Evaluation of role of admission test in intra partum patients admitted in labour ward in predicting adverse outcome of fetus at risk.

3. REVIEW OF LITERATURE

- **1960 MARSAC** a French physician was ridiculed in a poem by a colleague
- **PHILLIPE DE GOUST** claiming to hear the heart of the fetus "beating like the clapper of a mill"
- **LAENNAC** a physician working in Paris in 1806 was the father of the techniques of auscultation of adult heart and lungs.
- **LE JUMEAN** also a physician working with Leannac became interested in applying this technique to other conditions including pregnancy.
- It was not until **1818** that **FRANCOSIS ISSAC** Mayor at Geneva a surgeon reported the fetal heart audibility is different from the maternal pulse by applying the ear directly to the pregnant mother's abdomen.
- **JOHN CREESY FERGUSO** later to become first professor of medicine in 1827 was the person in Britain to describe the fetal heart sounds who subsequently published his famous work entitled observation on obstetric auscultation in 1833.

- **AURON FRIDRISH HOLIT** was the first to design the fetal stethoscope in 1834 be **BEPAUL** modified this although **PINARDS** name is most commonly associated with the stethoscope, his version followed several others only appearing in 1876.
- **WINKEL in 1893** empirically set the limits of normal heart rate at 120 – 160bpm.
- 1953. **GUNN** and **WOOD** reported the application and recording of fetal heart in the proceeding of the royal society of medicine.
- **In** 1958 **HON** pioneered electronic fetal monitoring in **USA CALDEYRO BARCIA** in Uruguay and **HAMACLIAS** in Germany reported their observation on the various heart rate patterns associated with fetal distress.
- The production of the first commercially available fetal monitor by **HAMMAELIER** and **HEWLELT PACKARD** in 1968 was soon followed by **SONICAID** in the U.S.A.
- **CALDEYRO BARCIA** and co workers in 1966 description is also widely known. Their type I dips correspond to those early uniform or variable deceleration or a combination of both and their type II dips correspond to late uniform decelerations.
- **KUBLI** et al 1969 graded variable deceleration according to amplitude and duration as mild, moderate and pronounced. Fetal

scalp blood PH correlated with this classification such that the mean PH with mild deceleration was 7.2 with moderate deceleration as 7.15.

- In 1968 the first clinical electronic fetal monitor became available and **PAUL ET AL** 1975 reported that such monitoring reduced both caesarean section for fetal distress and perinatal asphyxia.
- **TRIMBOS** and **KEIVSE** in 1978 performed 594 cardio tocograph records in all normal pregnancies between 34 – 40 wks of gestation and found no ominous pattern, 7.2 were suspicious and at least one such record was seen in 37% of all pregnancies. These results indicate the potential danger of false positive results in normal pregnancies if the method is used in appropriately.
- **SOLIUM** and Coworkers in 1979 – 1980 suggested classification into four groups was slightly modified by **MONTAN ET AL** in 1985. In practice 85 – 90% of all antepartum cardiotocograph records are normal, 6-8 are suspicious, 1-2 pathological.
- **KREBS ET AL** 1979 found frequency of low APGAR scores to be high 69.6% in the first 30 minutes of labour, when cardio tocograph was abnormal, compared with records that were normal 2.7%, suspicious 15.8%.

- In the Dublin fetal heart rate monitoring study **MAC DONALD ET AL** in 1985 compared continuous fetal heart monitoring with different auscultation. The neonatal seizures were significantly higher in the auscultation groups. 8.5% compared with the electronically monitored group2.4%.
- At 1985 In Kandang – Kerbau Hospital in Singapore an admission test was carried out on 1041 low risk patients. The trace is obtained for 20 minutes immediately on admission and it was sealed in an envelope and put aside for latter analysis. In this study women with ominous tracing 40% developed fetal distress and women with reactive tracing 1.4% developed fetal distress.
- Electronic fetal monitoring has been a subject of controversy for the last two decades. Several authors criticize for the policy of electronic fetal monitoring **(LEVENO ET AL** 1986 **SHY ET AL** 1990**)** claiming that it led to increase in caesarean section with no evidence of fetal benefits.
- A new test is required to pick up the apparently low risk women whose fetus is compromised on admission or likely to become compromised in labor. This is the admission test (**ARUL KUMARAN ET AL** and **GIBB** 1992).

- **ARULKUMARAN** with **GIBB** 1992, bearing the acute events the admission test may be good predictor of fetal condition at the time of admission and during the next few hours of labour in term fetuses labeled as low risk. It was estimated that in these situation for 50% of the babies, to become acidotic took 115 minutes with repeated variable declaration and 185 minutes with a flat trace. There fore it can be safely assumed that if the admission test was reactive it is reasonable to perform intermittent auscultation and 20 minutes of electronic monitoring 2-3 hourly in low risk labour. In high risk women (or) women with suspicious (or) abnormal admission tests should have continuous electronic fetal monitoring throughout labour.
- **INGEMARSSON ET AL** 1993, In low risk pregnancies fetal heart changes were found in 5-10% of antenatal records. A normal cardiotocograph record occur in only 50% of all labour. About 15% of all records have a base line abnormality with normal base line ranging from 110 – 150 BPM. 10% will have tachycardia, while frequency of bradycardia is less than 10%.
- **INGEMARSSON ET AL** 1993 the presence of acceleration is one key to reactive pattern on an antepartum cardio tocograph. The number of acceleration increased towards the end of the pregnancy

with the greater increase occurring between 28 and 34 weeks. Between 25 and 30 weeks of gestation deceleration are more common than acceleration in response to fetal movement. Most of the decelerations are of short duration (15 – 30S) with amplitude of 14-30 BPM. After 30 weeks accelerations are more common than decelerations in response to fetal movement. At term decelerations are not seen with fetal activities.

- Routine testing in high risk pregnancies has not shown to be of value and cardio tocograph should be regarded as a diagnostic tool rather that a screening test (**INGEMARSSON** 1933).
- The admission test cannot be expected to predict fetal distress that develops, several hours later in labour when the fetal condition was satisfactory at the time of admission. (**INGEMARSSON 1993**). It has a high predictive value for fetal well being 98.7% and high specificity but a rather predictive value of an abnormal test 40% and a low sensitivity 23.5%.
- **VINTZILEO ET AL** 1995 compared continuous electronic fetal heart rate monitoring versus intermittent auscultation and found electronic fetal heart monitoring is superior with better sensitivity, low specificity and higher positive and negative predictive values.

4. FETAL PHYSIOLOGY – CONTROL OF FETAL HEART RATE

In the fetus cardiac output and the O_2 supply to the brain are mainly heart rate dependent.

Control of the fetal heart:

Control of the fetal heart is a complex phenomenon. The fetal heart has its own intrinsic activity and a rate determined by the spontaneous activity of the pace maker. That is the sinoatrial node. This structure has the fastest rate and determines the rate of the normal heart. The next fastest pace maker is the atrium. AV node has the lowest rate of activity and generates ventricular rhythmn seen in complete heart block.

The fetal heart rate is modulated by a number of stimuli. CNS influence is important with cortical and sub cortical influence which is not under voluntary control. Other physiological factors regulate the heart rate such as circulatory catecholamines, chemoreceptors, baroreceptors, their interplay with the autonomic nervous system.

Baroreceptors

They are stretch receptors that are sensitive to changes in blood pressure and are situated in the arch of aorta and the aortic and the carotid sinus. In response to a rise in blood pressure impulses from the baro receptors are sent to cardioregulatory center, resulting in an increase in vagal stimulation. Thus heart rate is lowered in an attempt to restore the blood pressure to a normal level.

Chemo receptors:

They are situated in the carotid and aortic bodies in a similar position to that of the baroreceptors and also in the mid brain itself. The chemo receptors respond to changes in O_2 and CO_2 tension. A fall in O_2 in blood detected by the carotid and aortic bodies. This would result in an increased sympathetic discharge from the cardioregulatory centre. This causes an increase in FHR and thus blood pressure. If the fall in po_2 was severe then diversion of blood from the gut, liver and kidney to the vital organs, the brain and the heart would also result.

Adrenal Medullary Response:

In response to stress adrenal medulla releases the hormones noradrenaline, adrenaline which results in increase in fetal heart rate & force of cardiac contraction in a manner similar to sympathetic nervous stimulation.

Higher Centres in the Brain

They are responsible for the so called rest activity cycles. During the fetal rest cycle the fetus is apparently sleeping in utero with reduced fetal body and limb movements. In CTG, it manifests as absence of accelerations, reduced base line variability. A fetal rest cycle normally lasts for about 20minutes following which there is return to normal fetal movements and fetal heart rate variability.

Fetal Compensatory system in response to hypoxia

In response to hypoxia,

1) Stimulation of sympathetic nervous system and increased adrenal medullary activity results in an increased heart rate in an attempt to increase cardiac output and redistribution of blood flow to vital organs.

2) There is an increased break down of liver glycogen to supply energy to the fetus. As a result of this anaerobic metabolism there is an accumulation of lactic acid to produce a metabolic acidosis. Although initially the acidosis is compensated by fetal buffering system especially Hb, this will eventually be overcome and the acidosis will become more severe. When the PH drops below 7.0 enzyme systems are inhibited and if this is maintained for long time death will occur ultimately.

Factors that commonly cause hypoxemia:

1) Reduction in uterine blood flow

    i. Uterine hyper stimulation with oxytocin, (or) in association with abruptio placenta.

    ii. Fall in maternal blood pressure. (Eg) supine hypotension syndrome, hypovolaemic shock, epidural analgesia.

    iii. Placental insufficiency – secondary to hypertension.

2) Reduction in umbilical blood flow, compression of umbilical cord

5. ELECTRONIC FETAL HEART MONITORING BASIC AND CLINICAL SCENARIOS

Fetal Heart Rate trace has four easily definable features:

1) Base line Heart Rate, 2)Base line Variability 3)Acceleration

4) Deceleration

Base line Heart Rate:

- Base line fetal heart rate activity refers to the model characteristics that prevail apart from periodic acceleration (or) decelerations associated with uterine contractions.

- Base line fetal heart rate is identified by drawing a line through the mid point of the wriggliness which represents most commonly the rate after excluding acceleration and deceleration.

Normal

- Normal Base line Heart Rate of Term fetus is 110-150PM (FIGO guide lines 1987) At 16WKS -160/mt

  At term – 140/mt

Rate of decrease in fetal heart rate as age advances is 1 Beat/Week/Minute.

- Base line Heart rate below 110/mt that lasts for 15 minutes or longer.

  Moderate Bradycardia 100-110bpm, Severe Bradycardia <100bpm

- Mild bradycardia with good baseline variability may be benign and not an indication of hypoxia. It represents that mild hypoxia is being well compensated by the fetus.

- Severe bradycardia is a more serious prognostic sign and indicates that the fetus is failing to compensate. When associated with reduced (or) absent variability, hypoxia should be suspected with Heart Rate <100 bpm.

- When Heart rate is increased beyond 150 beats/mt lasting for more than 15 minutes.

  Moderate tachycardia 150-170bpm, Severe tachycardia >170bpm

Base line variability

- Is regulated largely by autonomic nervous system. Base line rate normally exhibits an oscillating form reflective of beat to beat

changes in rate. It gives the varying degrees of irregularity (or) variability when printed on graph paper. Such deviation in heart rate is defined as baseline variability. 2 types of base line variability are present they are short term, and long term.

- Short term variability reflects instantaneous change in fetal heart rate from one beat to the next. It is the measure of the time interval between each cardiac systoles.

- Long term variability reflects oscillatory changes that occur during the course of 1 minute and result in waviness of the baseline. Normal frequency is 3-5 cycles per minute.

- Measurement of BLV

Detected by assessing the band width of the wriggliness by drawing a line through the highest and lowest point in the wriggliness during any one cm segment of the trace. Preferably when trace is reactive (or) showing acceleration.

Normal 10-25bpm, Reduced 5-10bpm, Absent <5bpm

Periodic events:

- Periodic fetal heart rate events refer to deviations from baseline that are related to uterine contraction and fetal movement.

- They are i) Acceleration

  ii) Deceleration - Early, Late, Variable.

Acceleration:

- Accelerations are sporadic rise in FHR of>15 beats/mt from the base line lasting for 15 seconds (or) more.

- Normal reactive tracing should have at least of 2 accelerations with in 20 minutes period.

- Accelerations almost always confirm that the fetus is not acidotic at that time. It represents intact fetal neurohumoral, cardiovascular control mechanism linked to fetal behaviour states.

- Acceleration most commonly present in antepartum period and early labour in association with variable deceleration.

- Intrapartum accelerations are due to stimulation by uterine contraction , fetal movement , during fetal scalp blood sampling & during acoustic stimulation.

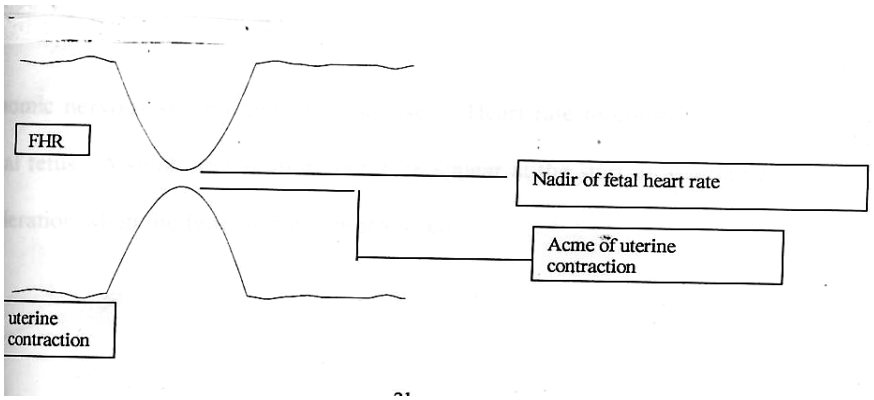
Absence of fetal heart acceleration during labour however is not necessarily an unfavorable sign unless co incidental with other reassuring changes.

Deceleration:

Drop in FHR by>15 beats/mt from the baseline lasting >15seconds

Early Deceleration

- They are synchronous with uterine contractions with a gradual decline and recovery, nadir of fall of fetal heart coincides with the acme of uterine contraction, mirroring the contraction.
- The drop in the heart rate is <40 beats/mt and it is due to head compression. Not due to fetal hypoxia or acidemia.
- Head compression leads to vagal nerve activation and heart rate deceleration. Most commonly seen during II stage of labour.



Variable Deceleration:

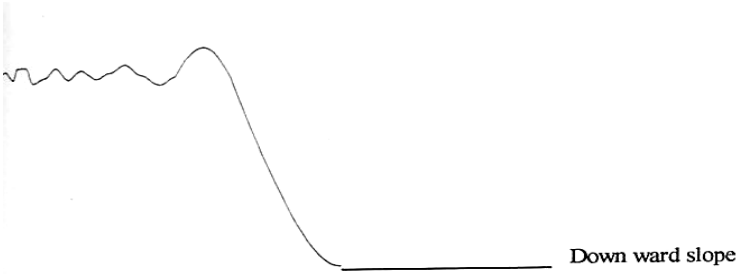
- Most common form seen in labour. They vary in occurrence in relation to contraction and also vary in shape and size. They show a precipitous fall and rise.
- Severe variable deceleration: when drop is>60 beats/mt and last for >60 seconds.
- It is due to umbilical cord compression.
- Most commonly seen in oligohydramnios, cord around the neck.

Mechanism of variable deceleration and its importance:

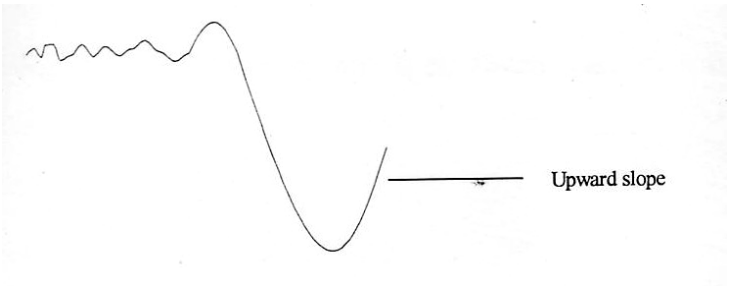
Umbilical vein has a thin wall and lower intraluminal pressure than the umbilical arteries. When compression occurs the blood flow through the umbilical vein is interrupted first before the artery. The fetus therefore loses

some of its blood volume. This will result in stimulation of autonomic nervous system and result in rise in heart rate to compensate in the normal fetus. A small acceleration therefore appears at the start of a variable deceleration when the fetus is not compromised.

Later umbilical arteries are also occluded and result in rise in systemic pressure in fetal circulation and the baroreceptors are stimulated. This results in fall in heart rate. This is responsible for the downward slope, when both vessels are occluded deceleration reaches nadir.



- During release of the cord compression arterial flow is restored first with a subsequent autonomicaly mediated sharp rise in heart rate. This is responsible for the upward slope.



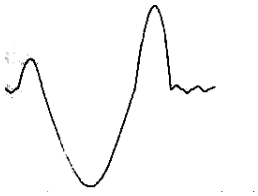
- Due to systemic hypotension blood is being pumped out culminating in a small acceleration after the deceleration.

- These accelerations before and after the deceleration are called shouldering. They are a manifestation of a fetus coping well with cord compression. Normal well grown fetus, can tolerate cord compression for a considerable length of time before they become hypoxic. Small growth restricted fetus already decompromised can’t with stand this cord compression and so it leads to hypoxia.

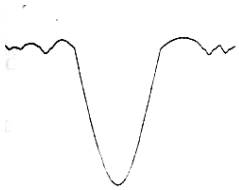
Various forms of variable deceleration and its significance

3) Normal shouldering – reassuring

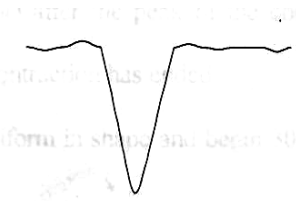
4) Exaggeration of shouldering – prepathological.



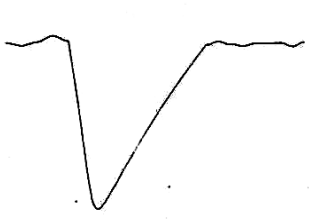
5) Loss of shouldering – pathological.



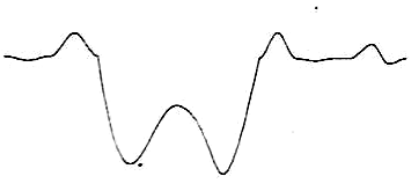
6) Smoothening of baseline variability within the deceleration which is associated with loss of variability at baseline and therefore pathological.



7) Late recovery – having the same pathological significance as the deceleration.



8) Biphasic deceleration same as late deceleration.



9) If duration of the deceleration is >60 seconds and the depth is >60 beats – it indicate progressive hypoxia.

Late deceleration:

Late deceleration is a symmetrical decrease in fetal heart rate beginning at (or) after the peak of the contraction and returning to baseline only after the contraction has ended.

- They are uniform in shape and begin 30 seconds or more after the onset of contraction.

- Nadir of deceleration is after the contraction acme.
- The return to baseline is well after the contraction is over.
- Descent and return of the fetal heart rate are gradual and smooth.
- Late deceleration not commonly associated with accelerations.

These late decelerations are due to uteroplacental insufficiency. The time interval (or) lag period from the onset of a contraction to the onset of a deceleration was directly related to basal fetal oxygenation. Length of the lagphase was predictive of the fetal PO_2. Slope and the amplitude of deceleration correlated with fetal oxygen tension.

Mechanism of late deceleration:

1. Chemoreceptor – mediated vagal reflex.
2. Direct hypoxic myocardial depression.

Clinical situations associated with late deceleration:

1. Maternal hypotension.
2. Excessive uterine activity.
3. Placental dysfunction.

Classification of intrapartum trace:

(A) Normal

Baseline rate 110-150bpm, Baseline variability 5-25bpm, Two accelerations in 20 minutes period and no deceleration

(B) Suspicious

Absence of acceleration (first to become apparent) for>40mts, Abnormal baseline rate 150-170bpm (or) 100-110bpm, Reduced baseline variability <10bpm and of greater significance if <5bpm and increased variability above 25 bpm, Variable deceleration without ominous features.

(C) Abnormal (or) ominous (or) pathological:

No acceleration +combination of the two following abnormal features.

1. Abnormal baseline rate and variability >90mts.(baseline below 100bpm (or) above 170 bpm and baseline variability <5bpm)
2. Repetitive late Decelerations.
3. Variable deceleration with ominous features duration >60s, late recovery, late deceleration component, poor baseline variability in between (or) during deceleration.

Other specific traces such as:

Sinusoidal pattern with out accelerations, Prolonged bradycardia <100bpm>10mts, Shallow deceleration in the presence of markedly reduced baseline variability <5bpm in a non reactive trace.

Significance of various tracing

Normal: Implies that the trace assures fetal health.

Suspicious: Indicates that continued observation (or) additional sample tests are required to ensure fetal health.

Pathological: Warrants action in the form of additional test or delivery depending on clinical picture.

Planning and management:

An admission test is helpful when planning the subsequent management of labour. High risk women (or) women with suspicious (or) abnormal admission test should have continuous electronic fetal heart rate monitoring throughout the labour.

A normal test is an insurance policy that permits us to encourage mobilization with no further need to perform electronic fetal monitoring for next 3-4 hours (or) until signs of the late first stage of labour are apparent.

CARDIOTOCOGRAPH – CLINICAL SCENARIOS

Causes for Bradycardia:

Head compression in case of occipito posterior (or) occipito transverse position, Congenital heart block., Serious fetal compromise like acute placental abruption, Maternal hypothermia – pyelonephritis, Under G.A for repair of cerebral aneurysm, Maternal cardio pulmonary bypass for open heart surgeries.

Causes for prolonged deceleration:

Uterine hyperactivity, Paracervical conductive analgesia, Cord prolapse, Uterine rupture, Placental abruption, Maternal hypoperfusion – supine hypotension syndrome. Haemorrhage due to trauma, Maternal hypoxia – eclampsia.

Causes for Tachycardia:

In preterm fetus due to early maturation of sympathetic system, Due to fetal movement, Increased sympathetic tone caused by arousal associated with noise, Fetal hypoxia – chronic, Hypovolemia – fetal, Anemia – fetal, Maternal dehydration, Administration of B- mimetic drugs to inhibit preterm labour and administration of atropine.

- Any baseline HR of >150 bpm should prompt a search for other suspicious features such as absent acceleration, poor baseline variability and deceleration.

- Gradually increasing hypoxia causes the FHR to rise gradually to a tachycardia. Steadily increasing baseline heart rate is very important.

- Provided in the presence of good baseline variability, acceleration and absence of deceleration, moderate bradycardia (or) tachycardia do not represent hypoxia.

Physiological causes for changes in BLV:

Fetal breathing, Fetal body movement, Advancing gestation. (upto 30 wks BLV is similar for both sleep and activity but after 30 WKS variability increased with activity).

Causes for decreased BLV:

Sleep (or) quiet phase of the FHR cycle, Hypoxia, Fetal acidemia – hypoxia, diabetic ketoacidosis, Prematurity, Tachycardia, Drugs- analgesics, sedatives, pethidine, phenothiazines, barbiturates, diazepam, antihypertensives acting on CNS, anaesthetics, GA, $Mgso_4$ Fentanyl, Local anaesthetics, Congenital

malformations of CNS, CVS, Cardiac arrhythmias, Fetal anaemia – rhesus disease, Fetal infection and Old machine without auto regulation

- Good variability by it self cannot be used as the only indicator of fetal well being.
- The good fetal heart variability alone should not be interpreted as necessarily reassuring.
- The development of decreased variability in the absence of deceleration is unlikely to be due to fetal hypoxia.
- Baseline Heart rate is fixed, less variable when heart rate is increased but BLHR is highly variable when heart rate is decreased.

Sinusoidal pattern:

Stable baseline – 110-150bpm., Regular oscillation 5-15bpm, Frequency 2-3 cycles/mt, Oscillation above or below the base line is equal, No area of normal fetal heart rate variability, no accelerations, Stimulation with vibro acoustic stimulation, produce acceleration, Commonly seen is fetal anemia (Eg) Rh Iso immunization.

Meconium stained amniotic fluid:

- In all cases with a fetal heart rate not belonging to normal category we have to release the amniotic fluid from above the presenting part. This is done by pushing the presenting part gently upwards. If no fluid, it indicates severe oligohydramnios and potential fetal compromise by cord compression during the process of delivery.

- If thick old meconium of brownish yellow colour liquor comes out, it indicates prolonged hypoxia due to placental insufficiency.

- If fresh meconium with cephalic presentation it indicates oligohydramnios, and if the trace is abnormal, immediate delivery is mandatory.

- Breech presentation with thick meconium is normal phenomenon.

- Acute stress such as placental abruption and umbilical cord prolapse paradoxically does not cause release of meconium.

- Presence of meconium in a preterm baby before 36wks indicates chronic listeriosis.

Twin Delivery:

- Twins are generally smaller than singleton due to IUGR. Second twin may be at greater risk than first twin and perinatal morbidity is high in multiple pregnancy. Hence continuous monitoring is necessary.

- If membranes have ruptured, 1st twin is best monitored by scalp electrode second twin monitored by external transducer. Both can also be monitored by external transducer with USG help.

- CTG monitoring is more useful in monitoring the second of the twin after the delivery of the first one. As long as CTG patterns are normal we can wait for progress. It is also helpful in intra uterine manipulation of the second twin.

Breech presentation:

In footling and flexed breech there are greater chances for cord compression and cord prolapse. They produce variable deceleration, sudden bradycardia in CTG tracing. The compression above the orbits by the uterine fundus is a mechanism for variable deceleration. It is very difficult to take blood sampling for PH in a breech presentation in labour and value may be different from that of scalp.

Eclampsia:

Convulsion represents a major stress to the fetus. After any major acute stress it is important to check fetal conditions by ultrasound (or) Doppler transducer of cardiotocograph before caesarean section.

Maternal drug effects:

Drug decreases the baseline variability with no deceleration, no acceleration and no increase in baseline rate. Drugs like azathioprine, cyclosporine, prednisolone, antibiotics, will cause reduced baseline activity. Other parameters will be normal except for reduced baseline activity.

Severe hypertension:

Abnormal cardio tocograph due to

i) Possible association with IUGR

ii) Medications like methyldopa reduces the baseline variability and acceleration.

iii) B- Blocker reduces baseline variability and acceleration.

Previous C.S:

Stability of the placental circulation and utero placental perfusion is dependent on the integrity of the uterus, and its vasculature. With dehiscence (or) rupture of the scar the major uterine blood vessels may become stretched

and torn compromising the perfusion of placenta. There is also possibility of umbilical cord prolapsing through the dehisced scar giving rise to a dramatic cord compression pattern.

Therefore changes in the FHR is the first sign of scar dehiscence

Epidural anaesthesia:

The insertion of anaesthetic agent in the epidural space can be associated with a degree of instability of the maternal vascular system. Providing the preceding trace has been normal, then this represents a stress to the fetus that it can with stand. After attention is paid to the circulating volume and vascular stability returns, then the trace returns to normal.

If the preceding trace has been abnormal then an ominous situation may develop, and birth by immediate C.S of compromised baby is mandatory.

Brow presentation:

Large mento vertical diameter presents at pelvic brim. Mechanical misfit leads to head compression and early and variable deceleration.

Face Presentation:

No special features with face presentation. Fetal electrode placement in face presentation should be avoided.

Second stage of labour:

Early decelerations are common in the second stage. Gradually they become deep and with variable features. Good recovery from each deceleration and a return to normal rate and variability however start before next contraction. Assisted delivery is necessary when signs of hypoxia present such as tachycardia (gradual), Reduced baseline variability in between and during deceleration, Late decelerations, Failure to return to normal after decelerations and Prolonged bradycardia.

3, 6, 9, &12 Minutes Rule for Bradycardia

At 3 minutes (FHR fails to return to normal and falls below 100) call the doctor.

At 6 Minutes ⟶ Prepare the mother

At 9 minutes ⟶ Prepare the forceps

At 12 minutes ⟶ deliver the baby

A delay of 20 minutes or more may result in an asphyxiated baby

Fetal conditions:

No acceleration , Reduced baseline variability, Baseline tachycardia in long standing hypoxia, Bradycardia in acute hypoxia

Anaemia:

Sinusoidal pattern, Without acceleration

Hydrocephalus:

Reduced baseline variability and low baseline rate.

6. ADMISSION TEST

With traditional assessment the fetal heart rate is auscultated after admission and every 15 minutes for a period of 1 minute after a contraction in the first stage of labor and after every 5 min in the second stage of labour. During auscultation the base line fetal heart rate can be measured but the other features of the FHR such as base line variability, acceleration deceleration are difficult to quantify.

Hence a new test is required to pickup the apparently low risk, high risk women whose fetus is compromised on admission or is likely to become compromised in labour. This is **Admission test.**

Admission Test:

It is a short, continuous electronic FHR recording made immediately on admission for a period of 20 minutes and gives a better impression of the fetal condition than the traditional assessment. An AT may identify those who are already at risk with an ominous pattern on admission even without any contractions. In those with normal or suspicious FHR, functional stress of the uterine contractions in early labour bring about the abnormal FHR changes. These changes may be subtle and difficult to identify by auscultation. Careful revision may reveal a reduced FHR.

In this Study:

Normal Trace

Recording with normal base line rate and variability, 2 acceleration of 15 beats above the base line for 15 seconds and no decelerations.

Suspicious (or) Equivocal Trace

No acceleration in addition to one abnormal feature such as reduced baseline variability <5, Presence of decelerations, Baseline tachycardia (or) bradycardia.

Ominous Trace

When more than one abnormal feature, Repeated ominous variable deceleration and Late deceleration.

To evaluate the outcome:

Fetal distress is considered to be present when ominous FHR changes led to caesarean section or forceps delivery or the newborn had an APGAR score < 7 at 5 minutes after spontaneous delivery.

Bearing the acute events, the AT may be good predictor of fetal condition at time of admission and during the next few hours of labour in term fetuses labelled as low risk.

Based on this continuous electronic monitoring for 20 minutes for every 2-3 hours and monitoring by auscultation in between can be recommended in low risk labour.

If the AT is normal and reactive a gradually developing hypoxia will be reflected by no acceleration and by gradually rising FHR. The latter can be picked up by the intermittent auscultation (or) electronic monitoring.

Well grown fetus with clear amniotic fluid with a reactive trace will take some time to develop an abnormal FHR pattern before acidosis develops.

In repeated late deceleration - 50 % of babies will take 115 minutes to become acidotic.

In repeated variable decelerations - 145 Minutes.

With a flat trace - 185 Mnutes.

Therefore it can be assumed that if the AT was reactive it is reasonable to perform intermittent auscultation every 15 minutes and 20 minutes of electronic monitoring every 2-3 hours in low risk patients.

Indications for electronic fetal monitoring

(A) Risk arising from maternal medical problems

Hypertension, Diabetes, Renal Disease, Collagen Disease, Severe anaemia, hemoglobinopathies, Cyanotic heart disease and Hyperthyroidism.

(B) Risk arising from problems of the fetus

IUGR, Post term, Pre term, Oligohydramnios, Multiple pregnancy, Breech presentation and Rh Iso Immunisation.

(C) Risk arising from problems of labour

Induced labour, Augmented labour, Prolonged labour, Prolonged rupture of membranes, Previous C.S, Regional analgesia, Antepartum (or) intrapartum vaginal bleeding and Intra uterine infection.

(D) Suspected fetal distress in labour

Meconium stained amniotic fluid, Abnormal suspicious admission test and Suspicious FHR on auscultation.

Planning Management

1. Admission test is helpful in planning the subsequent management of labour.
2. High risk women (or) women with suspicious or Ominous admission test should have continuous EFM throughout the labour.
3. Normal admission test is an insurance policy that permits us to encourage mobilization. Further need to perform EFM is after 3-4 hours (or) until evidence of late first stage of labour are apparent.

Duration of admission test

- Admission test should last as long as necessary until it is normal maximum up to 40 minutes.
- If two accelerations, normal rate and normal variability are seen in the first 5 minutes then that is very reassuring.

- If electronic fetal monitoring is commenced at the start of a quiescent phase for the fetus then it will need to continue until the fetus reawakes or the fetus can be stimulated by external noise and vibration or by artificial electronic pharynx. Most AT will last for 20 to 40 minutes.

7. MATERIALS AND METHODS

(A) Type of the Study **:** Cross Sectional Study.

(B) Cases **:** 100 Patients admitted in labour ward of both low risk and high risk were selected for the study.

Inclusion Criteria

Low risk cases:

1. Pregnant patients with gestational age of 37 weeks up to 40 weeks.
2. With labour pains either spontaneous (or) accelerated.
3. With cephalic presentation.

High risk cases :

Post dated pregnancy, Pregnancy induced hypertension, IUGR / Oligohydramnios, Rh negative pregnancy, Long period of primary infertility, Preterm labour, Bad Obstetric history, .Heart disease., Anaemia, Pr LSCS, Malpresentation.

Exclusion criteria

Antepartum haemorrhage, Multiple pregnancy, Major anomalies of fetus, GA of fetus <30 wks

C) Machine : Corometrics cardio tocograph fetal monitor

The FHR can be recorded using an external transducer placed on the maternal abdomen with the help of an abdominal belt. The external tocotransducer or tocodynamometer detects uterine activity by sensing a change in the tension of anterior abdominal wall. It consists of a central plunger coupled to a force transducer and outer guard ring. The plunger is pressed against the abdomen during the uterine contractions, which provides a qualitative assessment of the strength and frequency of uterine contractions.

D) Period : Period of 6 months labour ward posting in the period of 2 years

E) Place : Government Rajaji Hospital, Madurai

F) Method of the study :

- In this admission test was done for 100 patients in the labour ward at the time of admission.
- The patients were followed up according to the AT results.
- Patients with normal tracings were followed up by intermittent auscultation and electronic monitoring done once in 4 to 5 hours during monitoring. When we suspected fetal distress emergency intervention was made according to the stage of labour.
- In patients with suspicious, ominous tracings immediate ARM done and colour of the liquor was assessed. In patients with thin meconium stained amniotic fluid, amnio infusion was given and the labour was allowed to

progress. They were followed up carefully by intermittent auscultation and CTG monitoring. When there is change of colour of liquor or when ominous pattern appears on CTG record according to the stage of labour, the labour was terminated by either forceps or caesarean section. The findings of the admission test is correlated with the outcome of the pregnancy.

- To evaluate the outcome of pregnancy, fetal distress was considered to be present when ominous FHR changes led to caesarean section or forceps delivery and the new born had an APGAR score < 7 at 5 minutes following spontaneous delivery. (ARUL KUMARAN-GIBB).

G) p Value : The p value is calculated by computerized analysis of data utilizing the software Epidemiological information package 2002 (EPI Info 2002) developed by Centre for Disease Control and Prevention Atlanta for WHO.

8. RESULTS AND ANALYSIS

This study was conducted in Government Rajaji Hospital, Madurai for 100 patients who were admitted to labour ward. Admission test was performed to all these patients with Corometric Cardiotocograph machine. Fetal outcome was correlated with admission test findings. Of 100 cases, 50 were low risk, 50 were high risk cases. High risk factors being Post dated pregnancy, IUGR/Oligo hydramnios, Long period of primary infertility, Preterm labour, Bad Obstetric History, Heart Disease, Pregnancy induced Hypertension, RH negative, Anaemia, Previous LSCS, Face presentation.

Table 1

S.No.	Cases	Total Number	Percentage
1.	Low risk cases	50	50
2.	High risk cases	50	50
	Total	100	100
	Break up of High risk factors		
	a) Post Dated Pregnancy	17	34
	b) IUGR/Oligohydramnios	2	4
	c) Long period of infertility	4	8
	d) Pre term Labour	1	2
	e) Bad Obstetric History	4	8
	f) Heart Disease	3	6
	g) PIH	7	14
	h) Rh-ve	5	10
	i) Anemia	5	10
	j) Pr LSCS	1	2
	k) Face	1	2

This table shows various types of cases on whom admission test was performed. Among these low risk cases form 50% and high risk cases form 50%.

Table 2 : Age Wise Distribution

Age group	Total number	Percentage
18-24	68	68
25-29	27	27
30-34	3	3
35-39	2	2

Majority of them, 68% fall under 18-24 yrs. Remaining 27% of is formed by 25-29yrs age group persons. 3% is formed by 30-34yrs age group persons & 2% is formed by persons between 35-39yrs of age.

Table 3 : Obstetric Index

Gravidity	Total number	Percentage
Primi	70	70
G2	20	20
G3	9	9
G4	1	1

Among 100 patients, 70% of is constituted by the primis. Second gravida was 20%, Third gravida was 9% and Fourth gravida & above forms 1%.

Table 4 : CTG Tracing Pattern in all cases

CTG Pattern	Total number	Percentage
Normal Tracing	68	68
Suspicious Tracing	19	19
Ominous Pattern	13	13

Among 100cases, normal tracing was observed in 68% of cases, Suspicious tracing in 19% of cases & ominous tracing in 13% of cases.

Table 5 : CTG tracings in high risk cases

High risk cases	Normal	Suspicious	Ominous
PDP	11	3	3
IUGR/Oligo	-	-	2
Long pd	-	2	2
PTL	-	1	-
BOH	3	1	-
HD	2	1	-
PIH	4	3	-
Rh-ve	3	-	2
Anemia	2	2	1
Pr LSCS	-	1	-
Face	-	-	1

Among the high risk cases

25 cases had normal tracing, 14 cases had suspicious tracing, 11 cases had ominous tracing.

Table 6 : Mode of Delivery in all cases

Mode of delivery	Number	Percentage
Labour natural	61	61
Forceps delivery	17	17
Caesarean section	22	22

Among 100 patients, 6% delivered by Labour Natural, 17% delivered by Forceps, 22% delivered by Caesarean section

Table 7 : Mode of Delivery according to CTG findings

CTG Tracing	Mode of delivery		
	Labour natural	**Forceps delivery**	**Caesarean section**
Normal (n=68)	54	11	3
Suspicious (n=19)	5	6	8
Ominous (n=13)	2	-	11

In Normal tracings, Out of 68 cases, 54 delivered by labour natural 11 delivered of forceps. All the cases were delivered by forceps for non fetal distress indication. 3 cases delivered by LSCS were for fetal distress. Fetal distress may develop during the course of labour due to various reasons like hyper stimulation of uterus, short cord, cord around the neck & Intrapartum abruption of placenta. Among these 3 cases, all three developed fetal distress after 5 hours of AT. They needed emergency intervention inspite of normal tracing at admission according to stage of labour.

In suspicious pattern

Out of 19 cases;

5 delivered by labour natural. 6 delivered by forceps (4 cases for other than fetal distress indication, 2 cases for fetal distress as indication). 8 cases

were delivered by LSCS (3 cases for other than fetal distress indication, 5 cases for fetal distress as indication). So out of 19 cases, 14 cases required intervention either in the form of assisted vaginal delivery or LSCS.

In Ominous pattern

Out of 13 cases, 2 cases delivered by labour natural. 11 cases delivered by LSCS (5 cases for other than fetal distress indication, 6 cases for fetal distress as indication).

Table 8

Mode of delivery according to CTG Tracing in high risk cases

CTG Tracing	Mode of delivery		
	Labour natural	**Forceps delivery**	**Caesarean section**
Normal (n=25)	18 (72%)	5 (20%)	2 (8%)
Suspicious (n=14)	3 (21.4%)	4 (28.6)	7 (50%)
Ominous (n=11)	- (0%)	- (0%)	11 (100%)

Among high risk cases

In those with normal tracing (25), 18 (72%) Delivered by labour Natural, 5 (20%) Delivered by forceps, 2 (8%) Delivered by LSCS.

In those with suspicious tracing, 3(21.4%) delivered by labour natural, 4(28.6%) delivered by forceps, 7(50%) delivered by LSCS.

The patients were taken up for emergency LSCS in view of the high risk factors such as long period of infertility in(2) cases, PIH (1), Anaemia(1), Post dated pregnancy(1), Pr.LSCS(1), BOH (1).

In those with ominous tracing, 11 (100 %) were delivered by LSCS, to avoid fetal distress.

Table 9

Mode of delivery according to CTG Tracing in low risk cases

CTG Tracing	Mode of delivery		
	Labour natural	**Forceps delivery**	**Caesarean section**
Normal (n=43)	36 ((83.7%)	6 (14%)	1 (2.3%)
Suspicious (n=5)	2 (40%)	2 (40%)	1 (20%)
Ominous (n=2)	2 (100%)	- (0%)	- (0%)

Among low risk cases

In those with normal tracing, 36 (83.7% of) delivered by labour natural, 6 (14%) delivered by forceps for non fetal distress indication, 1 (2.3%) by LSCS. In this admission delivery interval was >6hrs.

In those with suspicious tracing, 2 (40%) delivered by labour natural, 2 (40%) delivered by forceps for non fetal distress indication, (1) 20% delivered by LSCS. In this case also admission delivery interval was more than 6hrs.

Table 10

Apgar Score according to CTG (all)

CTG pattern	Neonatal outcome (APGAR)		
	No asphyxia (7-10)	**Moderate asphyxia (6-4)**	**Severe Asphyxia (<4)**
Normal tracing (n=68)	65 (95.6%)	3 (4.4%)	- (0%)
Suspicious tracing (n=19)	11 ((57.9%)	7 (36.8%)	1 (5.3%)
Ominous tracing (n=13)	7 (53.8%)	2 (15.4%)	4 (30.8%)0

'p'=0.0004

There is statistically significant relationship ($p<0.05$) between CTG findings and Apgar score. Cases with normal CTG findings have high Apgar score. Suspicious and Ominous cases give birth to more children with moderate and severe asphyxia

Out of 100 cases, In those with normal tracings, 65(95.6%) developed no asphyxia, 3(4.4 %) developed moderate asphyxia. There were no cases of severe asphyxia.

In those with suspicious tracings, 11 (57.9 %) developed no asphyxia, 7 (36.8%) developed moderate asphyxia, 1 (5.3%) developed severe asphyxia.

In those with Ominous tracings, 7 (53.8%) developed no asphyxia, 2 (15.4%) developed moderate asphyxia, 4 (30.8%) developed severe asphyxia.

Table 11

Apgar Score according to CTG (in high risk group)

CTG pattern	Neonatal outcome (APGAR)		
	No asphyxia (7-10)	**Moderate asphyxia (6-4)**	**Severe Asphyxia (<4)**
Normal tracing (n=25)	24 (96%)	1 (4%)	- (0%)
Suspicious tracing (n=14)	9 (64.3%)	4 (28.6%)	1 (7.1%)
Ominous tracing (n=11)	7 (63.6%)	2 (18.2%)	2 (18.2%)

'p'=0.0133

The relationship between CTG pattern and Apgar scores is statistically significant ($p<0.05$) among high risk cases.

In High risk cases, In those with normal tracing, 24(96%) had no asphyxia, 1(4%) had asphyxia. This is attributable to the inherent risk factor in the high risk groups.

In those with suspicious tracing, 9(64.3%) had no asphyxia, 5(35.7%) had asphyxia.

In those with ominous tracing, 7(63.6%) had no asphyxia, 4(36.4%) had asphyxia.

Table 11A

Apgar Score according to CTG (in low risk group)

CTG pattern	Neonatal outcome (APGAR)		
	No asphyxia (7-10)	**Moderate asphyxia (6-4)**	**Severe Asphyxia (<4)**
Normal tracing (n=43)	41 (95.3%)	2 (4.7%)	-(0%)
Suspicious tracing (n=5)	2 (40%)	3 (60%)	-(0%)
Ominous tracing (n=2)	- (0%)	2 (100%)	2(100%)

'p'=0.0001

Apgar score and CTG findings are significantly related among low risk cases also.

In low risk cases, In those with Normal tracing, 41 (95.3%) had no asphyxia, 2 (4.7%) had asphyxia .This is attributable to the inherent risk factors of the process of labour.

In those with Suspicious tracing, 2(40%) had no asphyxia, 3(60%) had asphyxia.

In those with Ominous tracing, 4(100%) had asphyxia Apgar score or CTG findings are significantly related among low risk cases also.

Table 12

Results of AT in relation to the incidence of fetal distress

CTG Pattern	Admission test (n)	Fetal distress
Normal tracing	68	3 (4.4%)
Suspicious tracing	19	8 (42.1%)
Ominous tracing	13	5 (38.5%)

'p'=0.0001

There exists statistically significant relationship ($p<0.05$) between the results of AT and incidence of fetal distress among the total study cases.

In all cases, In those with Normal tracing, 3 (4.4%) developed fetal distress.

In those with Suspicious tracing, 8 (42.1%) developed fetal distress.

In those with Ominous tracing, 5 (38.5%) developed fetal distress.

Table 13

Results of AT in relation to the incidence of fetal distress in high risk group

CTG Pattern	Admission test (n)	Fetal distress
Normal tracing	25	1 (4%)
Suspicious tracing	14	5 (35.7%)
Ominous tracing	11	3 (27.3%)

'p'=0.0116

There exists statistically significant relationship ($p<0.05$) between the results of AT and incidence of fetal distress among the high risk cases.

In High risk group, In those with Normal tracing, 1(4%) developed fetal distress.

In those with Suspicious tracing, 5(35.7%) developed fetal distress.

In those with Ominous tracing, 3(27.3%) developed fetal distress.

Table 14

Results of AT in relation to the incidence of fetal distress in low risk group

CTG Pattern	Admission test (n)	Fetal distress
Normal tracing	43	2 (4.7%)
Suspicious tracing	5	3 (60%)
Ominous tracing	2	2 (100%)

'p'=0.0002

There exists statistically significant relationship ($p<0.05$) between the results of AT and incidence of fetal distress among the low risk cases.

With Normal tracings, 2 (4.7%) developed fetal distress, with Suspicious tracing, 3(60%) developed fetal distress, with Ominous tracing, 2(100%) developed fetal distress.

Table No. 15

Neonatal ICU admission

CTG Pattern	Total no. of cases	Admitted in ICU for asphyxia	Percentage
Normal tracing	68	-	0
Suspicious tracing	19	3	15.7
Ominous tracing	13	4	30.8

'p'=0.0006

The relationship between ICU admissions and CTG patterns is statistically significant among the total study cases.

In Normal Tracings, admission is nil. With suspicious tracing admission is 5.3%. With Ominous tracing admission is 30.8%.

Table No. 16

Neonatal ICU admission in high risk group

CTG Pattern	Total no. of cases	Admitted in ICU for asphyxia	Percentage
Normal tracing	25	-	0
Suspicious tracing	14	3	21.4
Ominous tracing	11	2	18.2

'p'=0.0251

The relationship between ICU admissions and CTG patterns is statistically significant among the high risk cases.

With normal tracing, admission is nil. With Suspicious tracing admission is 21.4%. With Ominous tracing admission is 18.2%.

Table 16A

Neonatal ICU admission in low risk group

CTG Pattern	Total no. of cases	Admitted in ICU for asphyxia	Percentage
Normal tracing	43	-	-
Suspicious tracing	5	-	-
Ominous tracing	2	2	100

'p'=0.0271

The relationship between ICU admissions and CTG patterns is statistically significant among the low risk cases also.

Table no. 17

Prediction of fetal distress

Screening test results	Fetal distress present	Fetal distress absent	Total
Positive (Abnormal CTG Pattern)	13(a)	19 (b)	32
Negative(Nnormal CTG)	3 (c)	65 (d)	68

'p'=0.00001

Fetal distress prediction results are significantly related to screening test findings ($p<0.05$) among total cases.

Sensitivity = True positive (a) / True positive (a) + false negative (c) x 100

= a / a+c x 100

= 13 / 16 x 100

= **81%**

Specificity = True Negative / False positive + True negative x 100

= d / b + d x 100

= 65 / 19 + 65 x 100

= **77%**

Positive predictive value = True positive / True positive + false positive x 100

= a / a x 100

$$= \frac{a}{a+b} \times 100 = \frac{13}{13+19} \times 100 = \mathbf{42\%}$$

$$\text{Negative predictive value} = \frac{\text{True negative}}{\text{False negative + true negative}} \times 100 = \frac{d}{c+d} \times 100 = \frac{65}{3+65} \times 100 = \mathbf{96\ \%}$$

Among total study cases, admission test in prediction of fetal distress has a Sensitivity of 81%, Specificity of 77%, Positive predictive value of 42% & Negative predictive value of 96%.

Table no. 17A

Prediction of fetal distress in high risk group

Screening test results	Fetal distress present	Fetal distress absent	Total
Positive (Abnormal CTG Pattern)	8 (a)	17 (d)	25
Negative (Normal CTG)	1 (c)	24 (b)	25

'p'=0.0232

Fetal distress prediction results are significantly related to screening test findings ($p<0.05$) among high risk cases.

Sensitivity = True positive (a) / True positive (a) + false negative (c) x 100

= a / a+c x 100

= 8 / 8+1 x 100

= **89%**

Specificity = True Negative / False positive + True negative x 100

= d / b + d x 100

= 24 / 17 + 24 x 100

= **59%**

Positive predictive value = True positive / True positive + false positive x 100

= a / a+b x 100

= 8 / 8+17 x 100

= **32%**

Negative predictive value = True negative x 100

False negative + true negative

= d x 100
d+c

= 24 x 100
1+24

= **96%**

In the high risk group, AT in prediction of fetal distress has a Sensitivity of 89%, Specificity of 59%, Positive predictive value of 32% & Negative predictive value of 96%.

Table no. 17B
Prediction of fetal distress in low risk group

Screening test results	Fetal distress present	Fetal distress absent	Total
Positive (Abnormal CTG Pattern)	5 (a)	2 (b)	7
Negative (Normal CTG)	2 (c)	41 (d)	43

'P'=0.0002

Fetal distress prediction results are significantly related to screening test findings ($p<0.05$)among low risk cases also ($p<0.05$).

Sensitivity = True positive (a) / True positive (a) + false negative (c) x 100

= a / a+c x 100

= 5 / 5+2 x 100

= 71%

Specificity = True Negative / False positive + True negative x 100

= d / b + d x 100

= 41 / 2+41 x 100

= 95%

Positive predictive value = True positive / True positive + false positive x 100

= a / a+b x 100

= 5 / 5+2 x 100

= 71%

Negative predictive value = True negative x 100

False negative + true negative

= d / d+c x 100

= 41 / 2+41 x 100

= **95%**

In low risk group, AT in prediction of fetal distress has a Sensitivity of 71%, Specificity of 95%, Positive predictive value of 71% & Negative predictive value of 95%.

	True positive (a)	**False positive (b)**	**False negative ©**	**True negative (d)**	**Sensitivity**	**Specificity**	**PPV**	**NPV**
a) High risk cases	8	17	1	24	89	59	32	96
b) Low risk cases	5	2	2	41	71	95	71	95
c) Total cases	13	19	3	65	81	77	42	96

9. DISCUSSION

In this study of 100 cases in the labour ward high risk cases were 50 in number and low risk cases were 50 in number. The high risk factors are postdated pregnancy, IUGR with oligohydramnios, long period of infertility, preterm labour, bad obstetric history, heart disease, pregnancy induced hypertension, Rh negative, Anaemia, Pr.LSCS and face presentation.

To reduce the likelihood of false diagnosis, tests of fetal well being must have a very high sensitivity at least 95%. (Thacker and Berkelman 1986).

Fetal activities are episodic (Campbell et al 1980, Dalton et al 1977, Patrick and Richardson 1985) and normal cycles can be up to 120 min. Manning FA, Platt LD, Sipos L Keegan KA have done Non stress test in High risk pregnancies American Journal of Obs. & Gyn. 135, 511-515.

Shime J, Gare DJ, Andrews J et al : Prolonged Preg : Surveillance of fetus and neonate and Course of Labour and delivery. Am J Obstet. Gyn. 148 : 547, 1984.

Weiner Z, Divon MY, Katz VK, et al : Multivariate analysis of antepartum fetal test in predicting neonatal outcome of growth retarded fetuses. Am J Obs. Gyn. 174.339, 1996.

In PIH; there is an increased risk of intrapartum hypoxia (Monton & Ingemarsson 1989).

FHR changes may be an earlier manifestation of loss of integrity of scar. (Beckley et al., 1991); (Arulkumaran et al, 1992) and prompt action should avoid fetal or maternal morbidity and mortality.

Abnormal FHR patterns suggest fetal hypoxia (Gibb and Arulkumaran, 1992); one should deliver these preterm fetuses early (Westgren et al; 1984).

Cooper RL, Goldenberg RL, Dubard MB, et al: Tocodynamometry and cervical length at 28 wks gestation. Prediction of spontaneous preterm birth. Am Obstet. Gyn. 172:666, 1995 Renu Misra 2004 - 95% reactivity of fetus at 34 wks.

In this study, about 68% fall among 18-24 yrs of age; 27% between 25-29 yrs, 3% between 30-34 yrs & 2% above 35yrs.Primis constitute 70%; second gravida about 20%; third gravida 9% and fourth gravida 1%. Normal tracing was seen in 68% of cases, suspicious tracing in 19% and ominous pattern in 13%. Ominous tracing was observed in 3 cases of postdated pregnancy, 2 cases of IUGR / Oligohydramnios, 2 cases of long period of infertility, 2 cases of Rh negative pregnancy, 1 case each of anaemia and face presentation.

Leveno K, J Quirk J. G, Cunninghan FG, et al : prolonged pregnancy and other high risk pregnancies. Observations concerning causes of fetal distress. Am J Obstet. Gyn., 150: 465, 1984.

Patients with no risk can also develop hypoxia and is not uncommon (Hobel et al, 1973, Ingemarsson, 1981; Arulkumaran et al, 1983).

Pillai M, James D (1990). The development of fetal heart rate patterns during normal low risk pregnancy. Obs. & Gyn. 76, 812-816.

On considering the mode of delivery, 61% delivered by labour natural, 17% delivered by forceps and 22% delivered by LSCS.

On considering the mode of delivery according to CTG findings with normal tracings 11 cases ended in assisted vaginal delivery for non fetal distress indication. LSCS was done in 3 cases which is due to the problems of the process of labour which is not predictable. 54 cases delivered by labour natural.

With suspicious trace; 6 cases ended up in assisted vaginal delivery out of which 2 cases were for fetal distress. 8 cases ended up in LSCS out of which 5 was done for fetal distress.

In ominous trace; 11 cases were taken up for immediate LSCS to prevent fetal hypoxia and poor Apgar score. 2 cases came late in labour and were allowed for vaginal delivery.

With normal tracings in high risk cases; 18 cases delivered by labour natural. All 5 cases were delivered by forceps for non fetal distress indication. 2 cases delivered by LSCS.

With ominous trace all 11 cases were taken up for LSCS bearing in mind the high risk factors inherent to the patients and also the tracing.

In those with suspicious trace, they were carefully monitored. 3 cases delivered by labour natural, 4 cases by forceps out of which 2 were for fetal distress and among 7 cases of LSCS 4 were for fetal distress as an indication. They were allowed to progress in labour and timely management was done.

In low risk cases with normal tracing only one case was taken up for LSCS with fetal distress as indication with admission delivery interval of >5 hrs .6 cases ended up in forceps but they were for non fetal distress indication 36 cases delivered by labour natural. In those with suspicious tracing, 2 delivered by labour natural, 2 by forceps which was for maternal exhaustion & 1 LSCS was done for fetal distress and admission delivery interval was >5 hrs. In those with ominous trace; 2 cases delivered by labour natural.

Although individual features of CTG are analyzed separately, normal or abnormal trends in these do not occur in isolation. So CTG should not only be evaluated quantitatively but recognition of an abnormal pattern qualitatively as a whole is important to judge the trace as normal or abnormal. (Renu Misra, Hans S Grundsell 2004). No abnormal CTG should be however ignored.

With normal tracing 95.6% of babies have no asphyxia. With suspicious trace 57.9% had no asphyxia. With ominous trace 53.8% had no asphyxia .

In high risk cases, with normal tracings 96% had no asphyxia. With suspicious tracing 64.3% had no asphyxia. With ominous tracing 63.6% had no asphyxia. In short, AT is 89% sensitive in high risk cases. In low risk cases, with normal

tracing 95.3% had no asphyxia. With suspicious tracing 40% had no asphyxia. In short AT is 95% specific in low risk cases.

Fresh thick meconium staining with scanty amniotic fluid may indicate hypoxia (Miller et al; 1975; Wong et al; 1985; Steer, 1985). The incidence of abnormal FHR patterns or hypoxia is more common with thick meconium stained amniotic fluid. (Meis et al; 1978; Arulkumaran et al; 1985; Steer 1985) found that incidence of low Apgar score was almost doubled if an abnormal FHR trace was present with thick meconium. With a normal FHR pattern incidence of low Apgar scores was not significantly increased. On comparing the incidence of fetal distress in normal tracing it was 4.4%.With suspicious tracing it was 42.1%. With ominous tracing it was 38.5%. In short normal tracing requires intermittent auscultation. Suspicious & Ominous traces require continuous electronic Fetal Heart Rate monitoring & timely intervention. In High risk group, there is a statistically significant relationship ($p<0.05$) between results of AT & incidence of fetal distress. It has Sensitivity of 89%, Specificity of 59%, Positive predictive value of 32%, and Negative predictive value of 96%. In low risk group also there is a significant relationship ($p<0.05$) between results of AT and incidence of fetal distress. It has Sensitivity 71%, Specificity 95%, Positive predictive value 71%, Negative predictive value 95%. Neonatal ICU admission with normal tracing nil. With suspicious tracing 5.3%. With

ominious tracing 30.8%. In high risk & low risk group the relationship between ICU admission & CTG pattern is statistically significant.

Arulkumaran S, Yeoh SC, Gibb DMF et al; obstetric outcome of meconium stained liquor, fetal distress in labours. Sing Med J, 532-526, 1985.

Low JA, Pancham SR, Worthington D, 1976. Fetal heart deceleration patterns in relation to asphyxia & fetal distress Obs. & Gyn; 47: 14-20.

Meis PJ, Hall M, Marshall JR et al; 1978. Meconium passage; a new classification of risk assessment during labour. Am J Obs. Gyn 11:509 - 513.

Millar FC, Sacks DA; Yeh SY et al; 1975 Significance meconium, fetal distress, asphyxia during labour. Am J Obs Gyn. 122: 573- 580.

Starks GC; 1980 Correlation of meconium stained amniotic fluid, Apgar scores are predictors of outcome Obs. Gyn., 56:604-605.

10. SUMMARY

In High risk cases, AT is more sensitive. In low risk cases, AT is more specific. Over all in both groups negative predictive value is 96%.

- AT is used to detect fetal well being and fetal distress if present at admission. This helps us in identifying the group of women who will require continuous electronic monitoring or intermittent auscultation during the course of labour.
- Antepartum risk factors are not accurate as predictors of fetal outcome. As fetal heart changes and acidosis occur in same frequency in high as well as low risk group during the course of labour.
- Bearing the acute events during the course of labour, AT will be a good predictor of fetal well being at the time of admission & during the next few hours of labour in term fetus.
- It will not predict the development of fetal distress that develops several hours later. (Ingemarsson 1993).
- Therefore it can be safely assumed that if the AT is normal it is enough to perform intermittent auscultation & CTG monitoring once in 4-5 hours. But abnormal tracings should have continuous monitoring through out labour to diagnose fetal distress earlier.

- LAVENO et al (1990) criticizes that the policy of continuous fetal monitoring led to increase in caesarean section with no evidence of fetal benefits.
- To improve the sensitivity and positive predictive value, false positives and false negatives are to be reduced. This can be done by doing additional tests like Fetal Scalp Blood Sampling (FSBS), Fetal Acoustic Stimulation Test (FAST); to diagnose exactly the fetal distress.

11. CONCLUSION

- Admission test is a good intrapartum test both in high risk & low risk groups. It is simple, highly acceptable and also it can be repeated.
- It has 89% sensitivity in high risk cases, 95% specificity in low risk cases and over all negative predictive value is 96%.
- A short recording immediately after admission can detect fetal distress if present & predict well being for next few hours.

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PROFORMA

Name : Date : Time:

Husband Name : Unit :

Age : IP No :

Type of Case : Booked ☐ Un booked ☐

Education : Wife : Husband :

Occupation : Wife : Husband :

Monthly family Income :

Socioeconomic history :

Class I ☐ Class II ☐ Class III ☐ Class IV ☐ Class V ☐

Habitation : Urban ☐ Rural ☐

History of Present illness :

Past History :

Menstrual history :

Married Since :

Obstetric table : G P L A LCB

High Risk Factors:

1. Post dated pregnancy ☐
2. IUGR / Oligohydramnios ☐
3. Long period of primary infertility ☐
4. Preterm labour ☐
5. Bad Obstetric History ☐

6. Heart disease ☐

7. Pregnancy Induced Hypertension ☐

8. Rh Negative pregnancy ☐

9. Anaemia complicating pregnancy ☐

10. Pr.LSCS ☐

11. Face ☐

Clinical examinations:

1. Ht. [ ] cm Wt. [ ] Kg.

2. Findings at time of admission:

G.E :

P/A :

FH :

P/V :

Admission test:

Normal ☐ Suspicious ☐ Ominous ☐

Type of Labour : Accelerated ☐ Spontaneous ☐

Date and Time of Delivery

Admission Delivery Interval in hours

Mode of Delivery

Labour Natural ☐

Forceps Delivery LSCS

For fetal distress ☐ For fetal distress ☐

Non fetal distress ☐ Non fetal distress ☐

Complications During labour

Fetal		Maternal	
Meconium stained liquor	☐	Abruptio placenta	☐
Cord around the neck	☐	Rupture of uterus	☐
Short cord	☐		
Cord Prolapse	☐		

Outcome of pregnancy :

Live birth	☐	Intra partum death	☐

APGAR

1 Min.	☐	5 Min.	☐

Birth weight in Kg :

Congenital Malformation if any

Yes ☐ No ☐

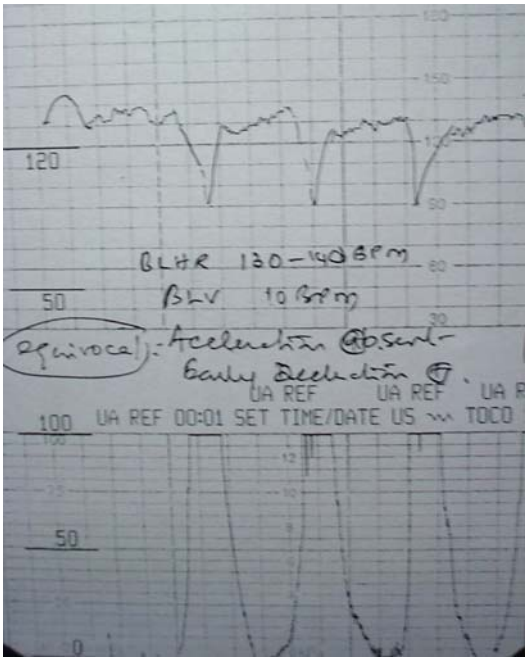
Sex of the baby : Male ☐ Female ☐

Neonatal death : Yes ☐ No ☐

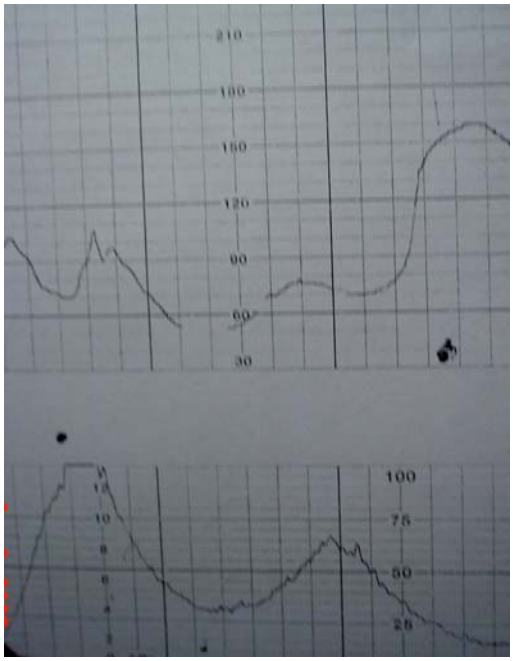
Cause :

Date :

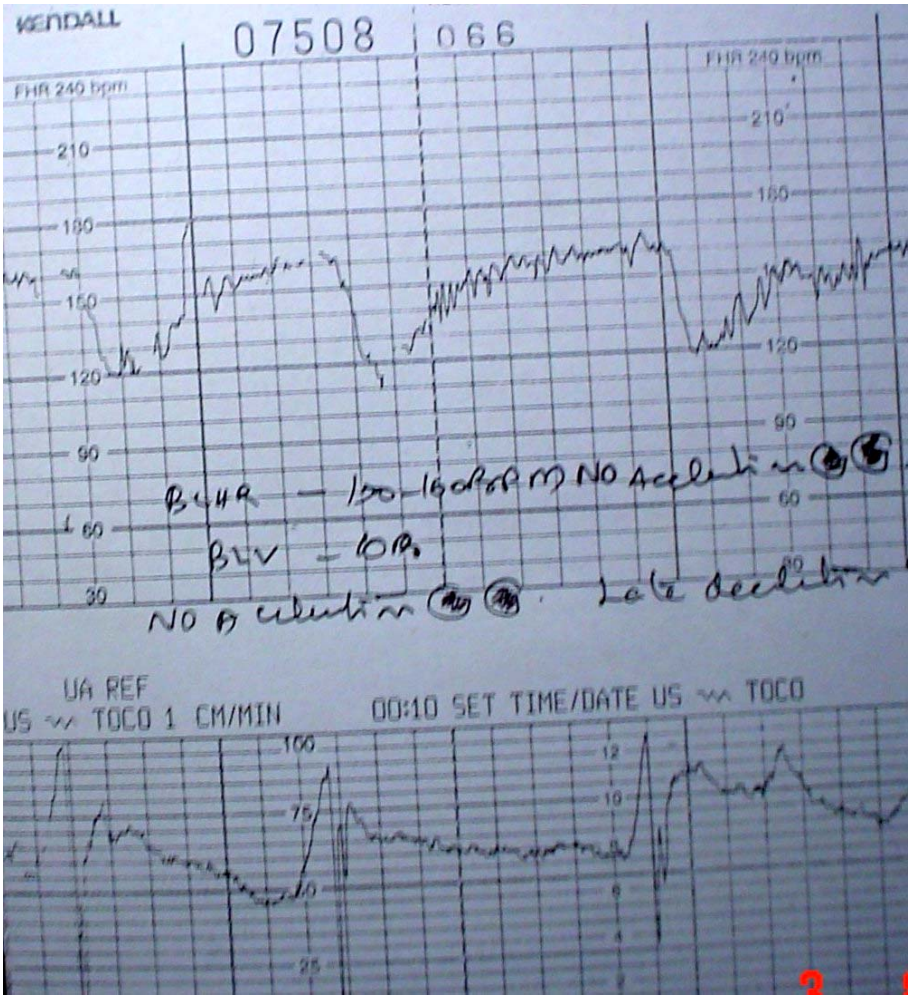
EARLY DECELERATION



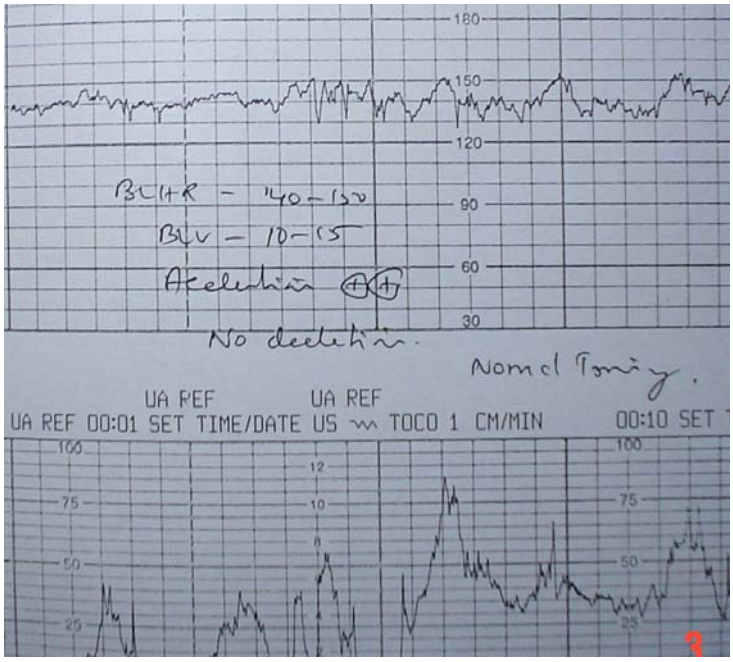
VARIABLE DECELERATION



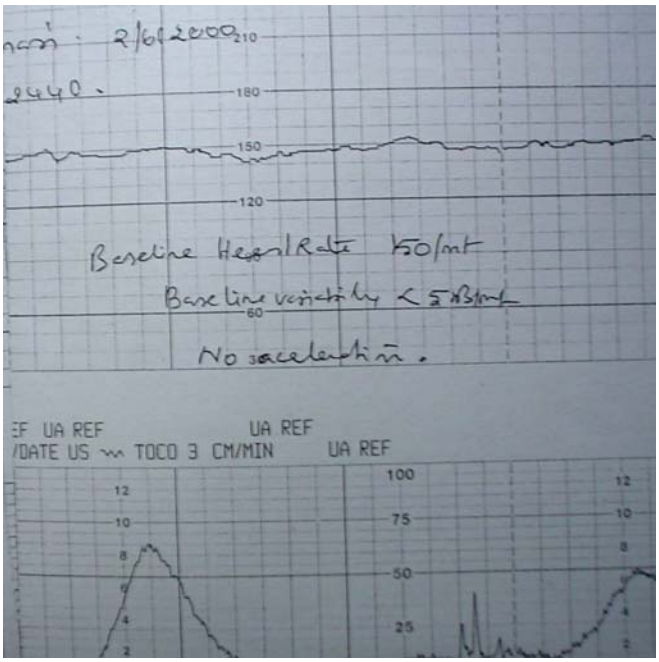
LATE DECELERATION



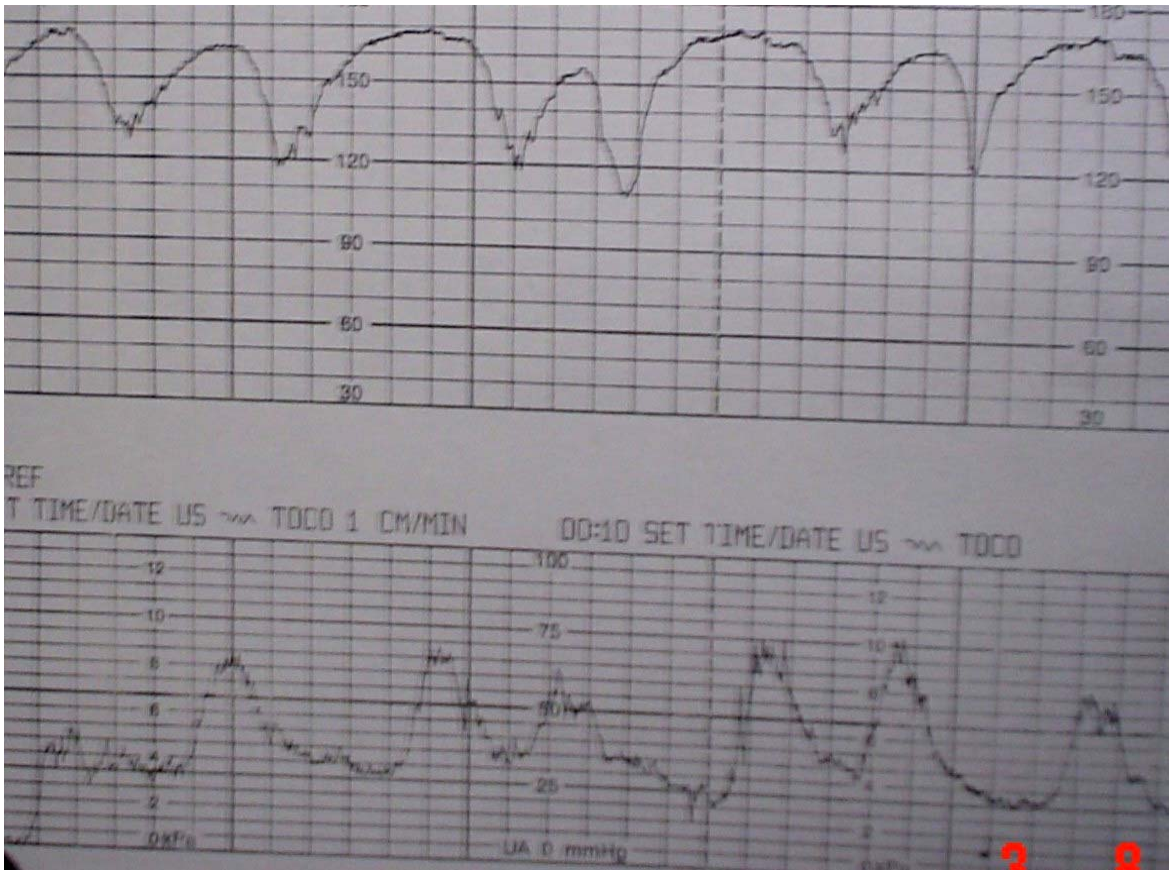
NORMAL TRACING



SUSPICIOUS TRACING

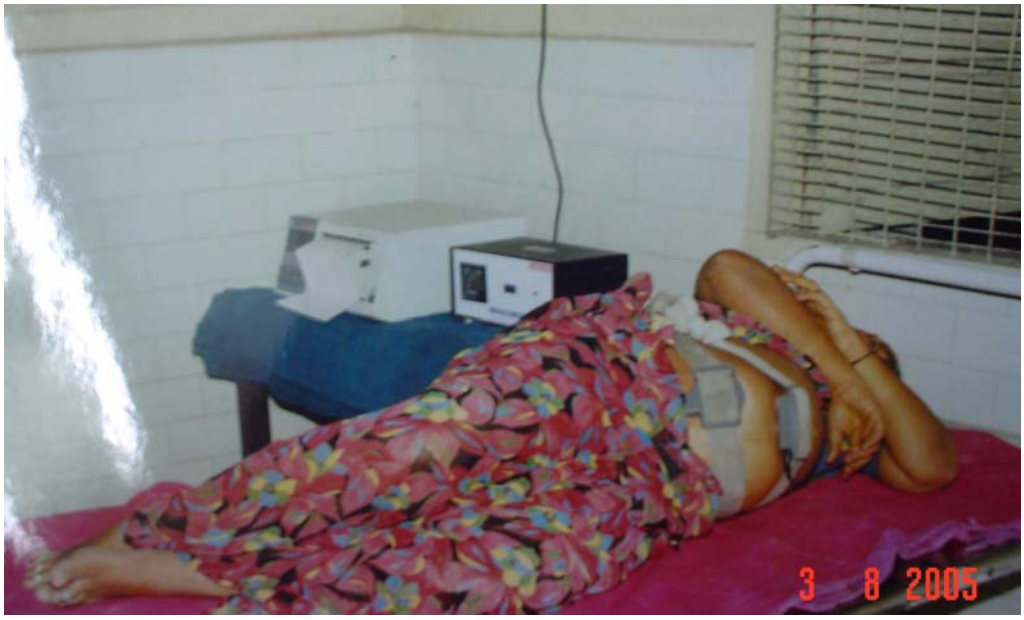


OMINOUS TRACING



CARDIO TOCOGRAPH – FETAL MONITOR

ADMISSION TEST



S.No	Age	Obstetric Index	Type of case	CTG Pattern	Mode of delivery	Admission Delivery Interval	Birth Weight (in Kgs.)	Sex	Apgar		Intrapartum complications	Follow up
1	22	Primi	HR	Ominous	LSCS	72hrs	3	F	7/10	9/10	-	-
2	22	Primi	HR	Ominous	LSCS	36hrs	1.75	F	7/10	9/10	-	-
3	27	Primi	HR	Ominous	LSCS	40hrs	3	M	7/10	9/10	-	-
4	25	Primi	HR	Ominous	LSCS	20hrs	3	M	7/10	9/10	-	-
5	25	G3P2L1	HR	Suspicious	LN	72hrs	2	F	7/10	9/10	-	-
6	21	G2 A1	HR	Normal	LN	33hrs	3	F	7/10	9/10	-	-
7	23	Primi	HR	Normal	LN	33hrs	2.1	M	7/10	9/10	-	-
8	25	G2P1L1	HR	Normal	LN	12hrs	2.75	M	7/10	9/10	-	-
9	21	Primi	HR	Ominous	LSCS	48hrs	2.5	M	7/10	9/10	-	-
10	24	Primi	HR	Normal	LN	19hrs	2.8	F	7/10	9/10	-	-
11	20	Primi	HR	Normal	LN	9hrs	2.75	M	7/10	9/10	-	-
12	24	Primi	HR	Normal	LSCS	14hrs	2.75	M	7/10	9/10	Thin meconium stained liquor	-
13	30	G2P1L1	HR	Normal	LN	36hrs	2.6	M	7/10	9/10	-	-
14	23	Primi	HR	Normal	LN	31hrs	3.25	M	7/10	9/10	-	-
15	29	Primi	HR	Ominous	LSCS	10hrs	3.4	M	7/10	9/10	Cord once round neck	-
16	24	G2P1L1	HR	Ominous	LN	6hrs	2.75	M	4/10	9/10	Thin meconium stained liquor	Observation
17	24	G3P2L2	HR	Normal	LN	6hrs	3	M	7/10	9/10	-	-
18	19	primi	HR	Normal	LN	43hrs	2.1	F	7/10	9/10	-	-
19	21	Primi	HR	Suspicious	LN	23 1/2 hrs	3	F	7/10	9/10	-	-
20	21	Primi	HR	Normal	Forceps	3hrs	3.25	F	7/10	9/10	-	-
21	22	G2P1L1	HR	Suspicious	LN	8hrs	2.9	F	7/10	9/10	-	-
22	29	G2P1L1	HR	Normal	LN	48hrs	3.1	M	7/10	9/10	-	-
23	27	G2A1	HR	Normal	Forceps	26 1/2hrs	2.9	M	7/10	9/10	-	-
24	22	Primi	HR	Suspicious	Forceps	4 1/2hrs	2.7	F	7/10	9/10	Short cord	-
25	24	Primi	HR	Normal	LN	4 1/2hrs	3	M	7/10	9/10	-	-
26	28	Primi	HR	Suspicious	LSCS	18 1/2hrs	3.7	M	4/10	6/10	Thick meconium stained liquor	Admitted in NICU

27	25	Primi	HR	Ominous	LSCS	8 1/2hrs	3	F	3/10	4/10	Thick meconium stained liquor	Admitted in NICU
28	21	Primi	HR	Ominous	LSCS	16hrs	3.25	F	7/10	9/10	Cord round the neck	-
29	25	Primi	HR	Suspicious	LSCS	78hrs	2.3	F	7/10	9/10	-	-
30	19	Primi	HR	Normal	LSCS	5days	2.7	M	7/10	9/10	-	-
31	22	G3P1L1A1	HR	Ominous	LSCS	17hrs	3.1	M	5/10	6/10	Thick meconium stained liquor	-
32	22	Primi	HR	Suspicious	LSCS	5 days	2.8	M	7/10	9/10	-	-
33	26	Primi	HR	Suspicious	LSCS	21hrs	2.5	F	7/10	9/10	-	-
34	25	Primi	HR	Suspicious	LSCS	8 1/2hrs	3.1	F	4/10	6/10	Thick meconium stained liquor	Admitted in NICU
35	22	G2P1L1	HR	Suspicious	LSCS	14hrs	2.8	M	7/10	9/10	-	-
36	19	G2A1	HR	Suspicious	LSCS	3 1/2hrs	2.4	F	7/10	9/10	-	-
37	28	G3P2L1	HR	Ominous	LSCS	4hrs	3.1	M	3/10	4/10	Thick meconium stained liquor	Admitted in NICU
38	22	Primi	HR	Suspicious	Forceps	28hrs	2.75	M	5/10	6/10	Thin meconium stained liquor	-
39	19	Primi	HR	Normal	LN	17hrs	3	F	7/10	9/10	-	-
40	19	G2A1	HR	Normal	LN	43hrs	3.25	F	7/10	9/10	-	-
41	32	G3A2	HR	Normal	LN	42hrs	3	M	5/10	6/10	Thin meconium stained liquor	-
42	21	Primi	HR	Suspicious	Forceps	7hrs	3.4	M	5/10	6/10	Thin meconium stained liquor	-
43	22	G2A1	HR	Normal	LN	10 1/2hrs	3.25	M	7/10	9/10	-	-
44	27	G3P2L1	HR	Normal	LN	48hrs	1.7	F	7/10	9/10	-	-
45	19	Primi	HR	Normal	LN	13 1/2hrs	2.3	F	7/10	9/10	-	-
46	19	Primi	HR	Normal	Forceps	4 hrs	2.5	M	7/10	9/10	-	-
47	27	G2P1L1	HR	Normal	Forceps	7 1/2hrs	2.7	F	7/10	9/10	-	-
48	21	Primi	HR	Suspicious	Forceps	12 hrs	2.75	M	3/10	4/10	Cord twice round the neck	Admitted in NICU
49	19	G3A2	HR	Normal	LN	24hrs	2.5	F	7/10	9/10	-	-
50	22	G2A1	HR	Normal	Forceps	81hrs	2.5	M	7/10	9/10	-	-
51	18	Primi	LR	Normal	LN	48hrs	2.4	F	7/10	9/10	-	-
51	24	Primi	LR	Normal	LSCS	13hrs	2.75	M	7/10	9/10	-	-
53	21	Primi	LR	Normal	LN	6hrs	3.2	F	7/10	9/10	-	-
54	23	Primi	LR	Normal	LN	17hrs	2.4	F	7/10	9/10	-	-

55	20	G2P1L1	LR	Suspicious	Forceps	10hrs	2.8	F	4/10	6/10	-	-
56	23	Primi	LR	Normal	LN	40hrs	2.25	F	7/10	9/10	-	-
57	22	Primi	LR	Normal	LN	7hrs	2.7	F	7/10	9/10	-	-
58	21	Primi	LR	Normal	LN	13 1/2hrs	2.75	M	7/10	9/10	-	-
59	23	G3P2L2	LR	Normal	LN	13hrs	2.7	M	7/10	9/10	-	-
60	32	G2P1L1	LR	Normal	LN	2 1/2hrs	2.7	F	7/10	9/10	-	-
61	25	G2P1L1	LR	Normal	LN	12hrs	2.7	M	7/10	9/10	-	-
62	20	Primi	LR	Normal	LN	4hrs	2.5	M	7/10	9/10	-	-
63	27	G2P1L1	LR	Suspicious	LSCS	8 1/2hrs	2.5	M	7/10	9/10	-	-
64	19	Primi	LR	Normal	Forceps	21 1/2hrs	2.75	F	4/10	6/10	Cord round neck	-
65	19	Primi	LR	Normal	LN	2hrs	2.9	M	7/10	9/10	-	-
66	27	Primi	LR	Normal	LN	5hrs	2.7	M	7/10	9/10	-	-
67	22	Primi	LR	Normal	LN	4hrs	3.2	F	7/10	9/10	-	-
68	35	G3P2L1	LR	Normal	LN	15hrs	2.5	F	7/10	9/10	-	-
69	30	G3P2L1	LR	Normal	LN	2hrs	2.2	F	7/10	9/10	-	-
70	35	G3P2L2	LR	Normal	LN	3hrs	3.1	M	7/10	9/10	-	-
71	20	Primi	LR	Suspicious	Forceps	36hrs	2.75	M	7/10	9/10	-	-
72	20	Primi	LR	Normal	LN	12hrs	3	M	7/10	9/10	-	-
73	25	Primi	LR	Ominous	LN	2hrs	2.5	M	2/10	3/10	ord twice round neck with thick meconi	-
74	20	G2P1L1	LR	Normal	LN	37hrs	2.9	F	7/10	9/10	-	-
75	20	Primi	LR	Normal	LN	3hrs	2.2	M	7/10	9/10	-	-
76	27	G4P3L3	LR	Normal	LN	17hrs	2.2	F	7/10	9/10	-	-
77	20	Primi	LR	Suspicious	LN	20hrs	2.25	F	5/10	6/10	Cord round neck	-
78	27	G2P1L1	LR	Normal	LN	27hrs	2.1	F	7/10	9/10	-	-
79	25	Primi	LR	Normal	LN	12hrs	3.3	F	7/10	9/10	-	-
80	28	Primi	LR	Normal	LN	26hrs	2	M	4/10	6/10	Hyperstimulation of uterus	-
81	20	Primi	LR	Normal	LN	15hrs	2.5	M	7/10	9/10	-	-
82	20	Primi	LR	Normal	LN	12hrs	2.75	M	7/10	9/10	-	-

83	22	Primi	LR	Ominous	LN	1hr	1.5	F	2/10	4/10	Cord once round the neck	-
84	23	Primi	LR	Normal	Forceps	8hrs	3.2	M	7/10	9/10	-	-
85	25	Primi	LR	Normal	LN	2hrs	2	M	7/10	9/10	-	-
86	23	Primi	LR	Normal	LN	19hrs	2.5	F	7/10	9/10	-	-
87	25	Primi	LR	Normal	Forceps	2hrs	2.8	M	7/10	9/10	-	-
88	19	Primi	LR	Normal	LN	3hrs	2.7	M	7/10	9/10	-	-
89	21	Primi	LR	Normal	LN	23hrs	3.4	F	7/10	9/10	-	-
90	20	Primi	LR	Normal	LN	72hrs	2.5	F	7/10	9/10	-	-
91	19	Primi	LR	Normal	LN	1 1/2hrs	3.4	F	7/10	9/10	-	-
92	24	Primi	LR	Normal	LN	3 1/2hrs	2.2	F	7/10	9/10	-	-
93	24	Primi	LR	Normal	LN	3hrs	2	F	7/10	9/10	-	-
94	19	Primi	LR	Normal	LN	3 1/2hrs	2	F	7/10	9/10	-	-
95	22	Primi	LR	Normal	Forceps	8hrs	3	M	7/10	9/10	-	-
96	20	Primi	LR	Normal	Forceps	53hrs	2.5	F	7/10	9/10	-	-
97	21	Primi	LR	Suspicious	LN	6hrs	2.5	F	4/10	6/10	Thick meconium	-
98	19	Primi	LR	Normal	LN	2hrs	2	F	7/10	9/10	-	-
99	20	Primi	LR	Normal	LN	36hrs	2.5	M	7/10	9/10	-	-
100	24	G3P2L2	LR	Normal	LN	1 1/2hrs	2.75	M	7/10	9/10	-	-

Neonatal death if any
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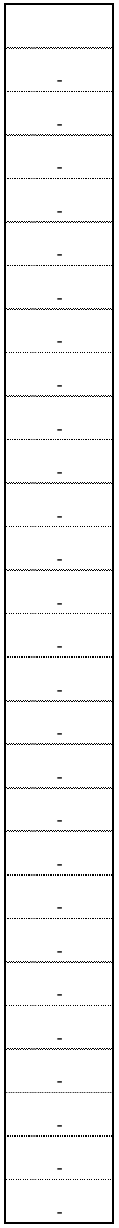
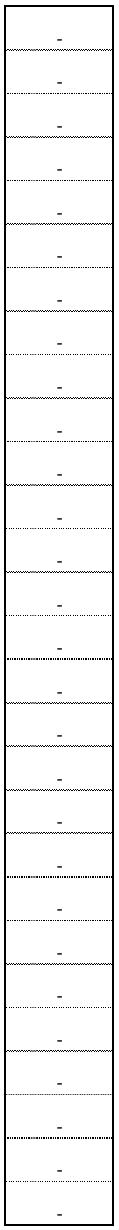
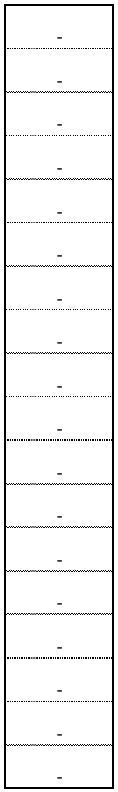
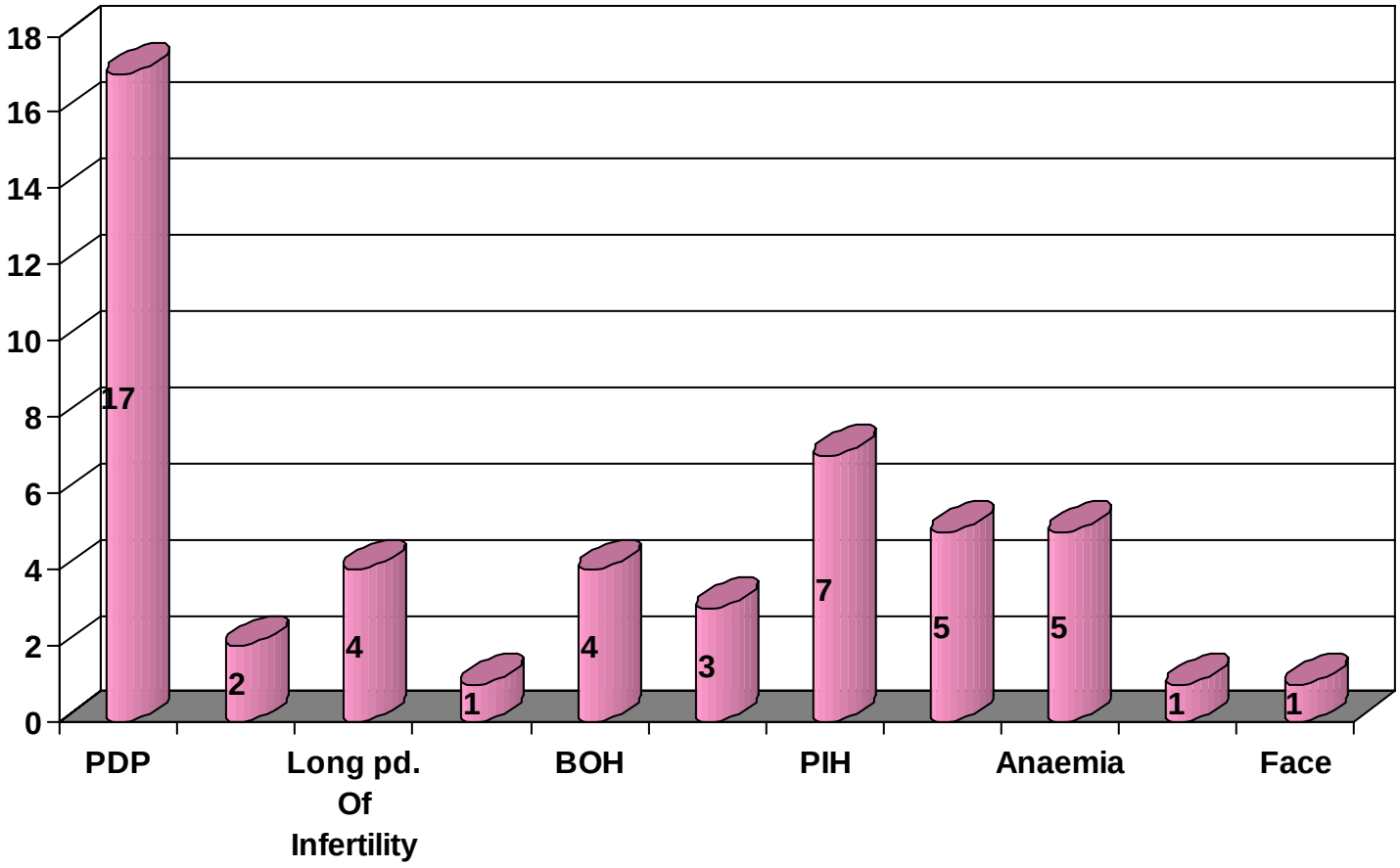


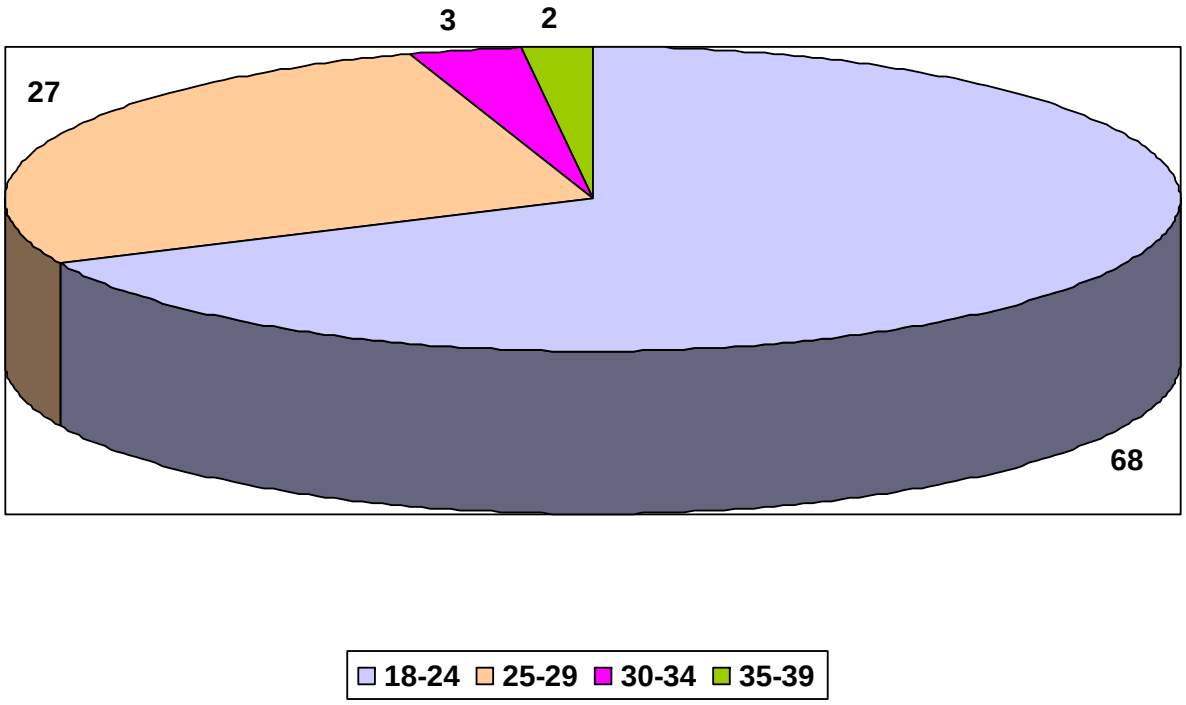




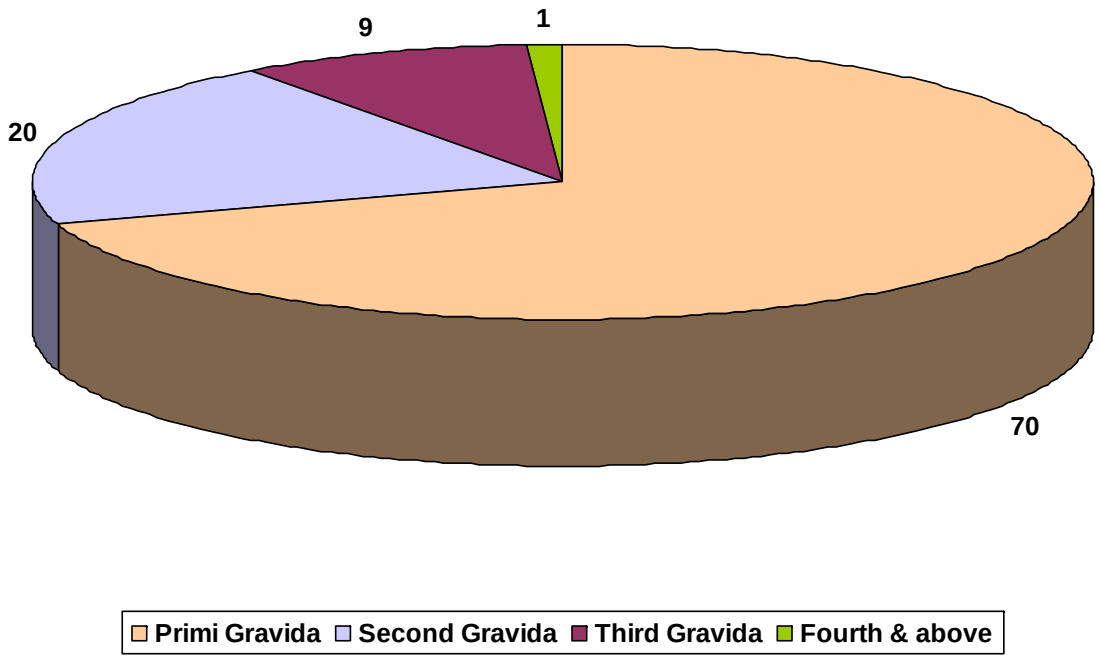
High Risk Cases



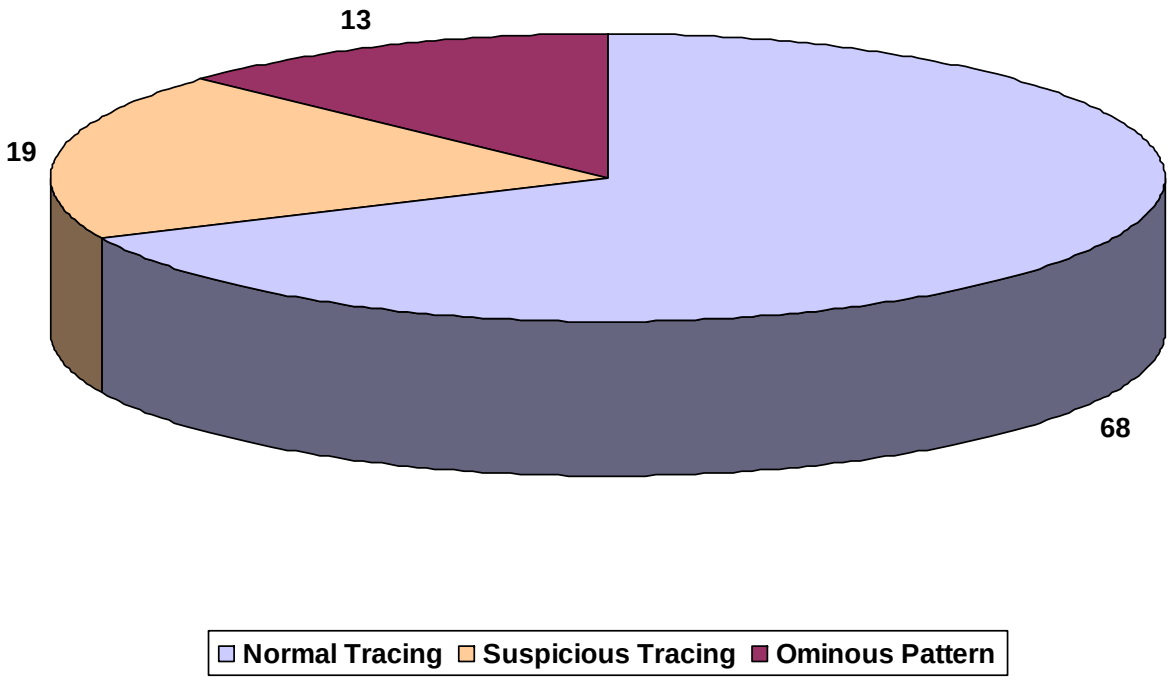
Age wise distribution



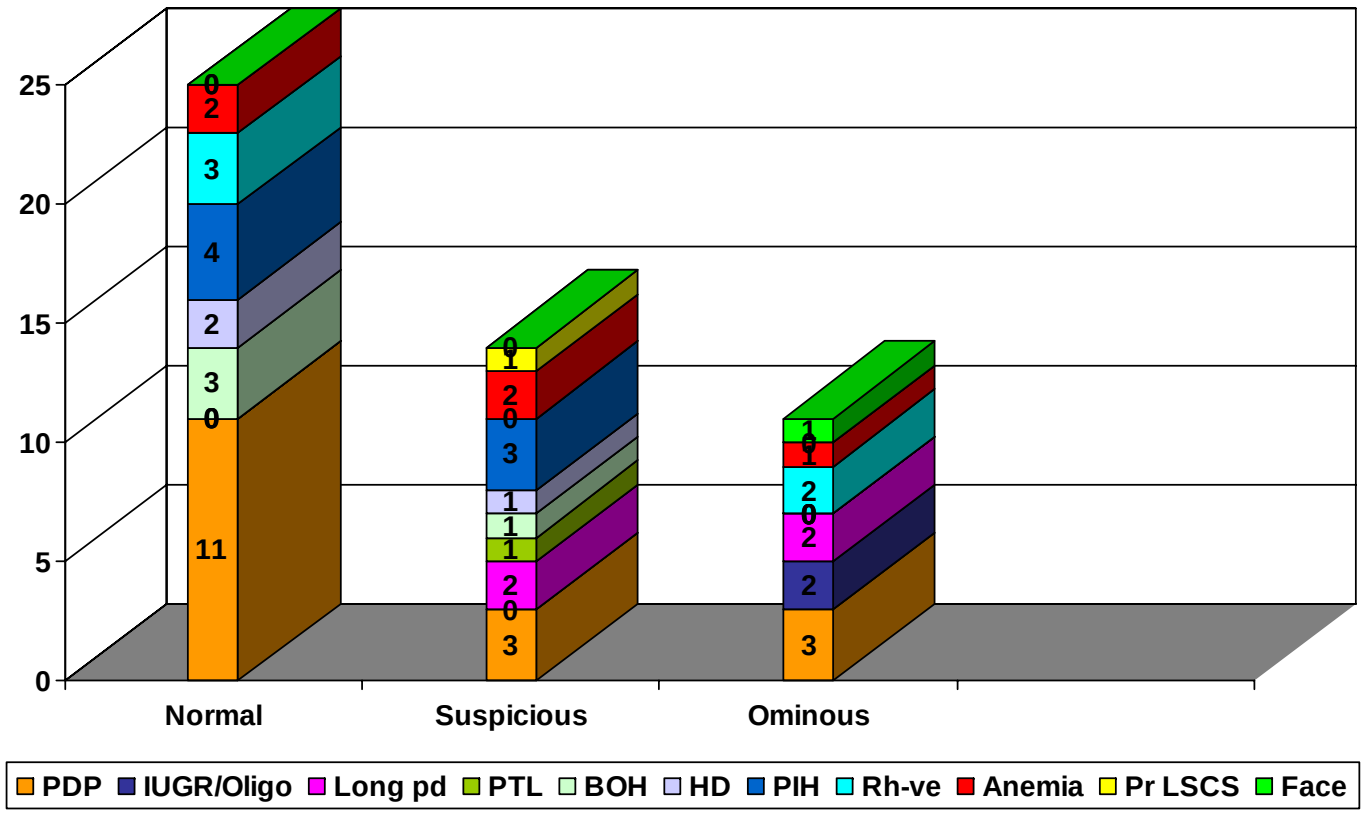
Obstetric Index



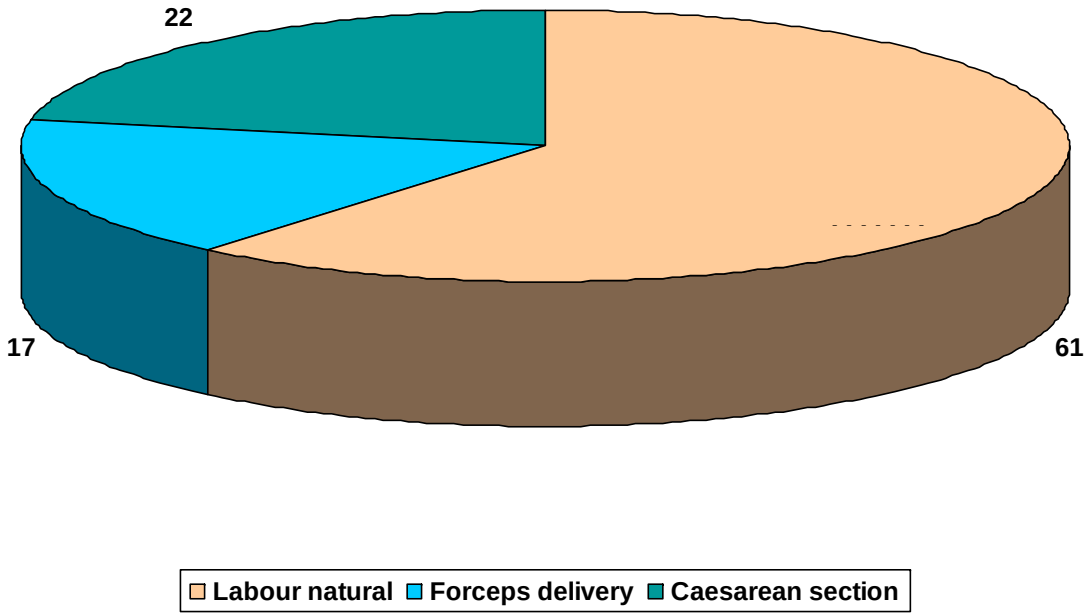
CTG Tracing Pattern



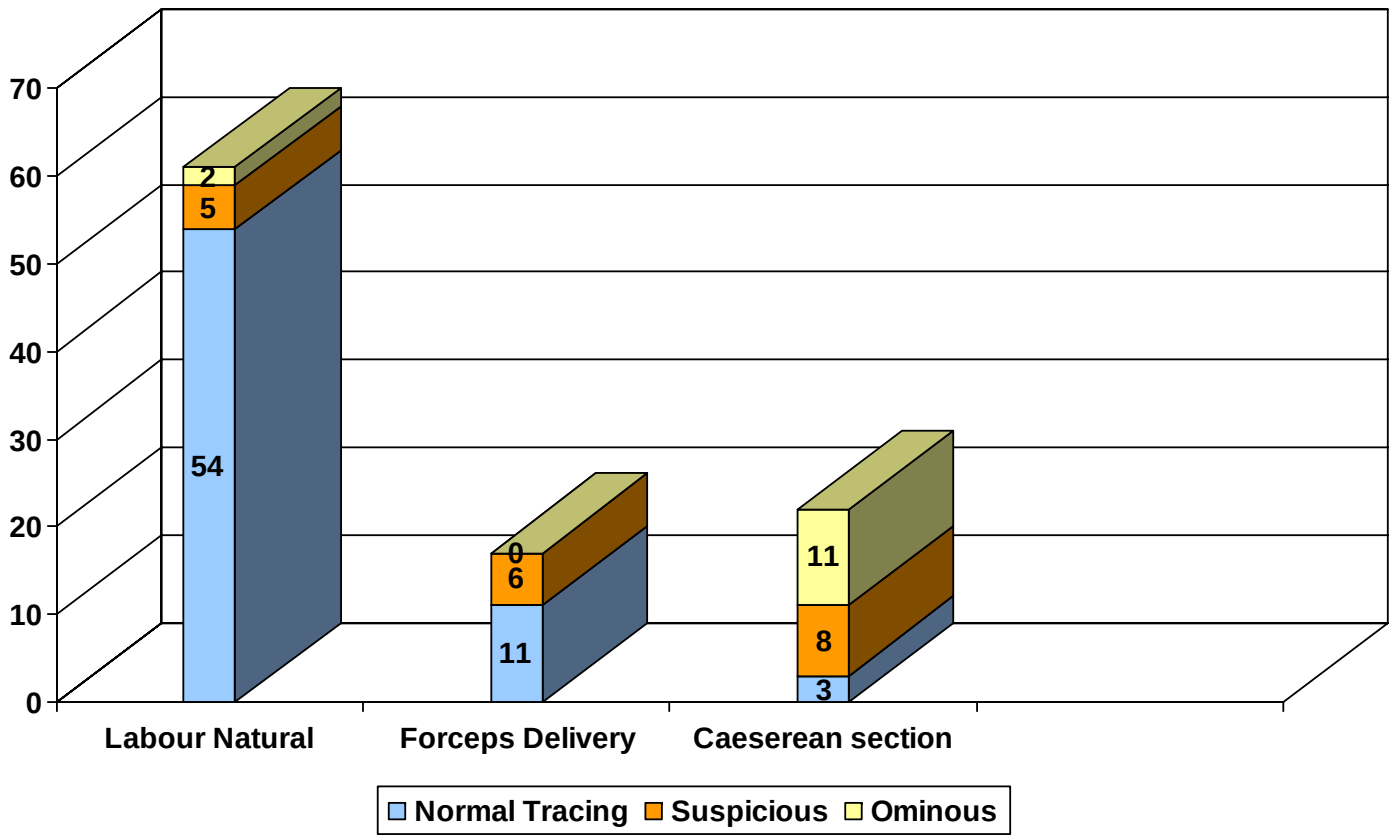
CTC tracings in High Risk Cases



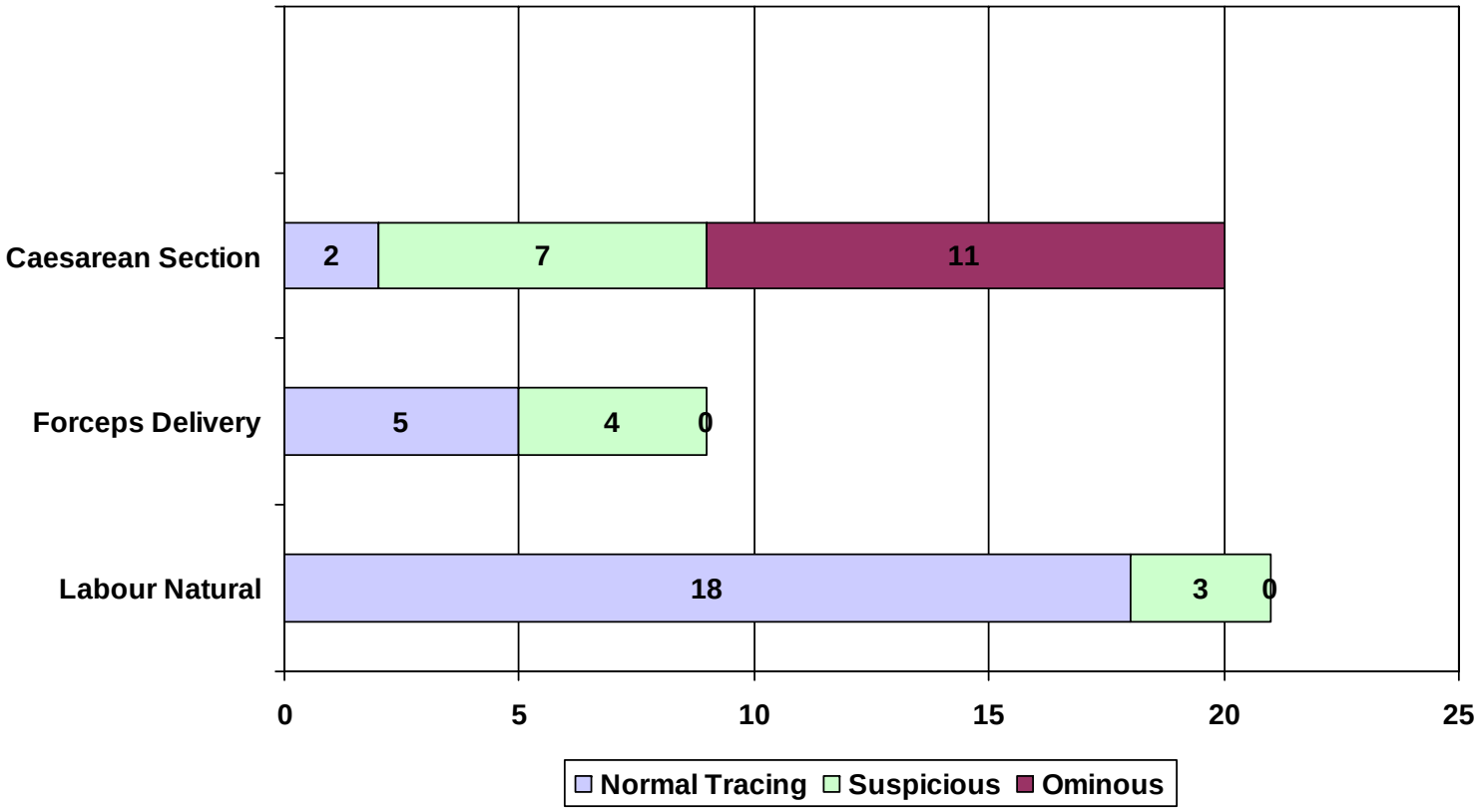
Mode of Delivery



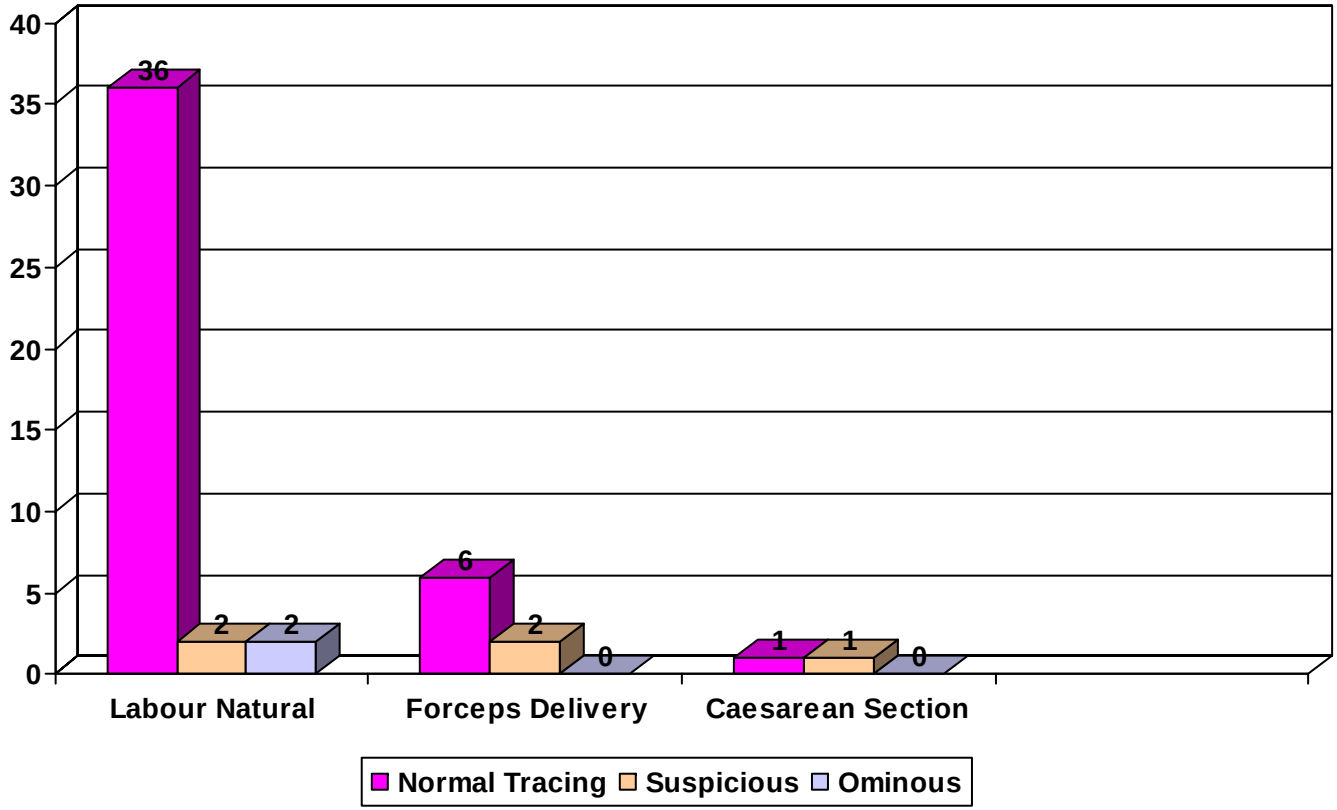
Mode of Delivery according to CTG findings



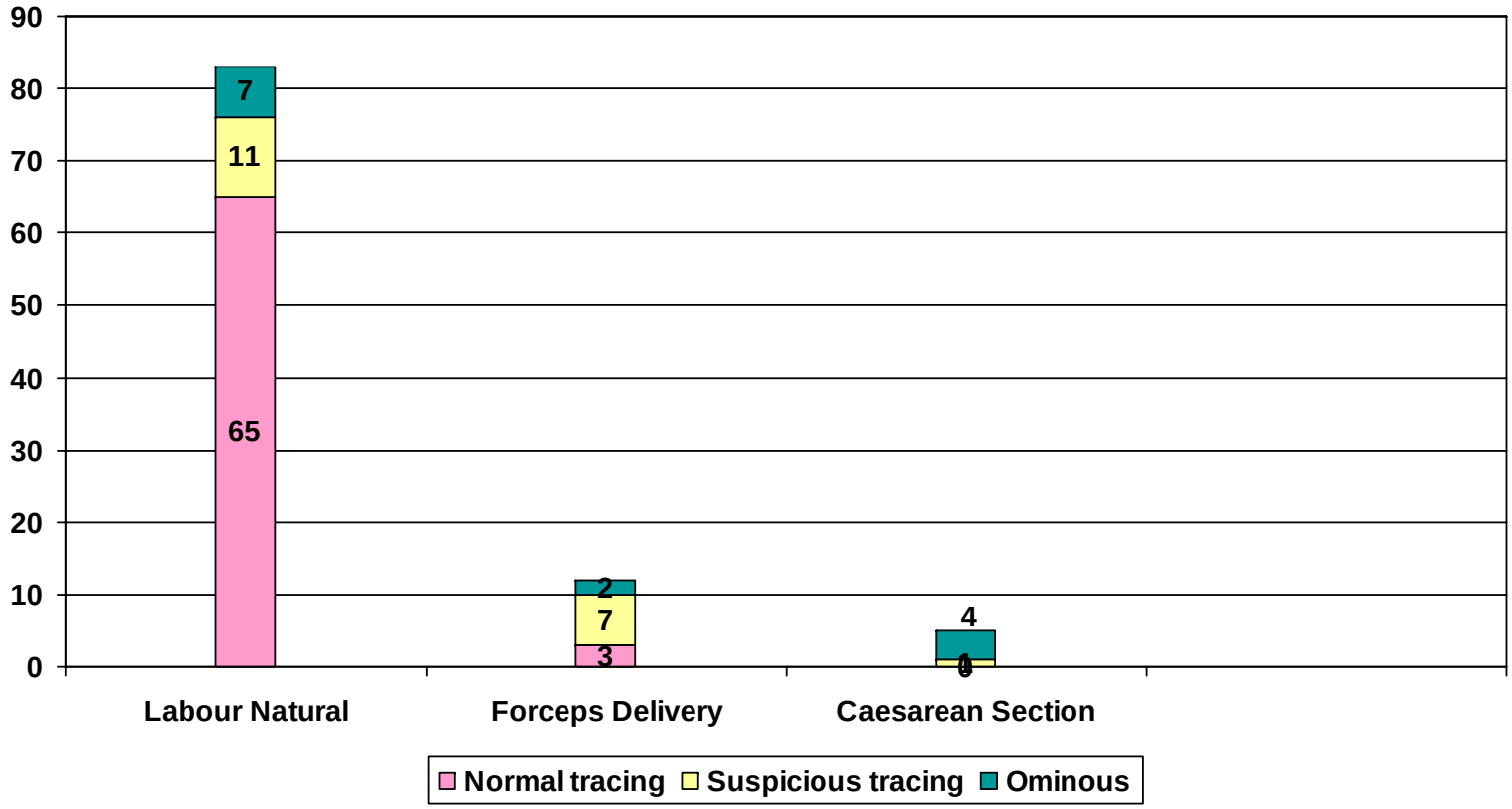
Mode of Delivery according to CTG findings in High Risk Cases



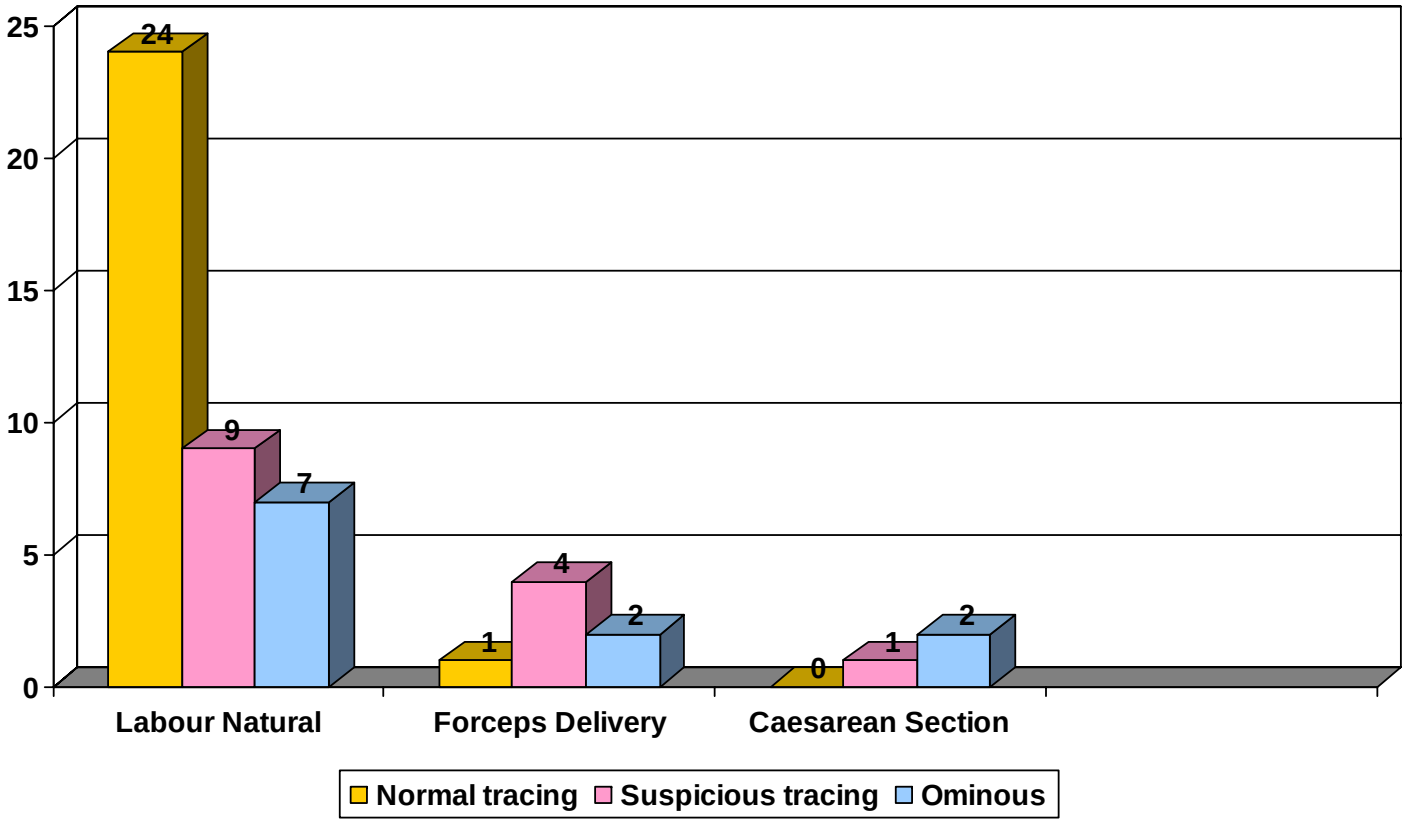
Mode of Delivery according to CTG findings in Low Risk Cases



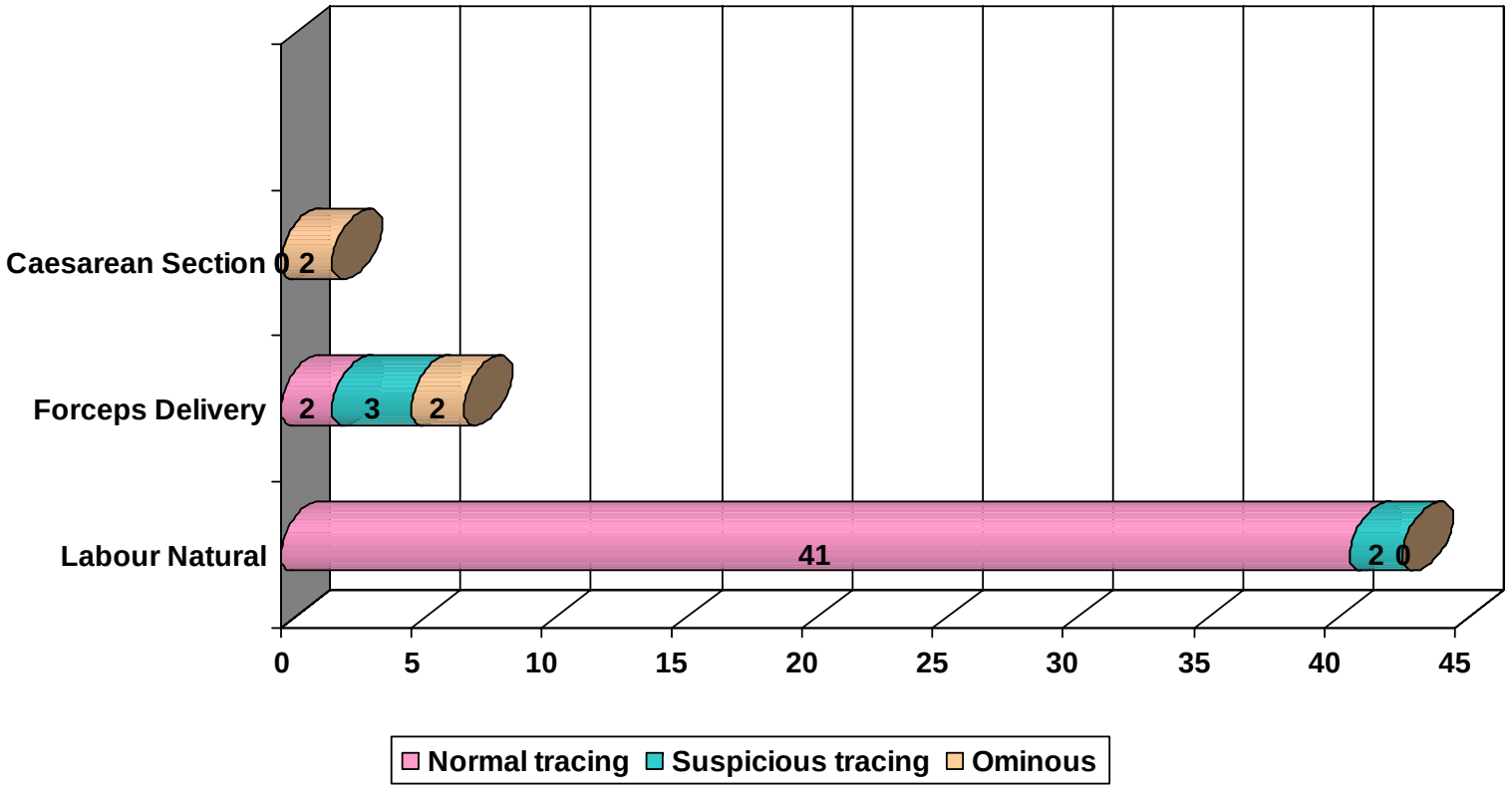
Apgar Score according to CTG pattern in all cases



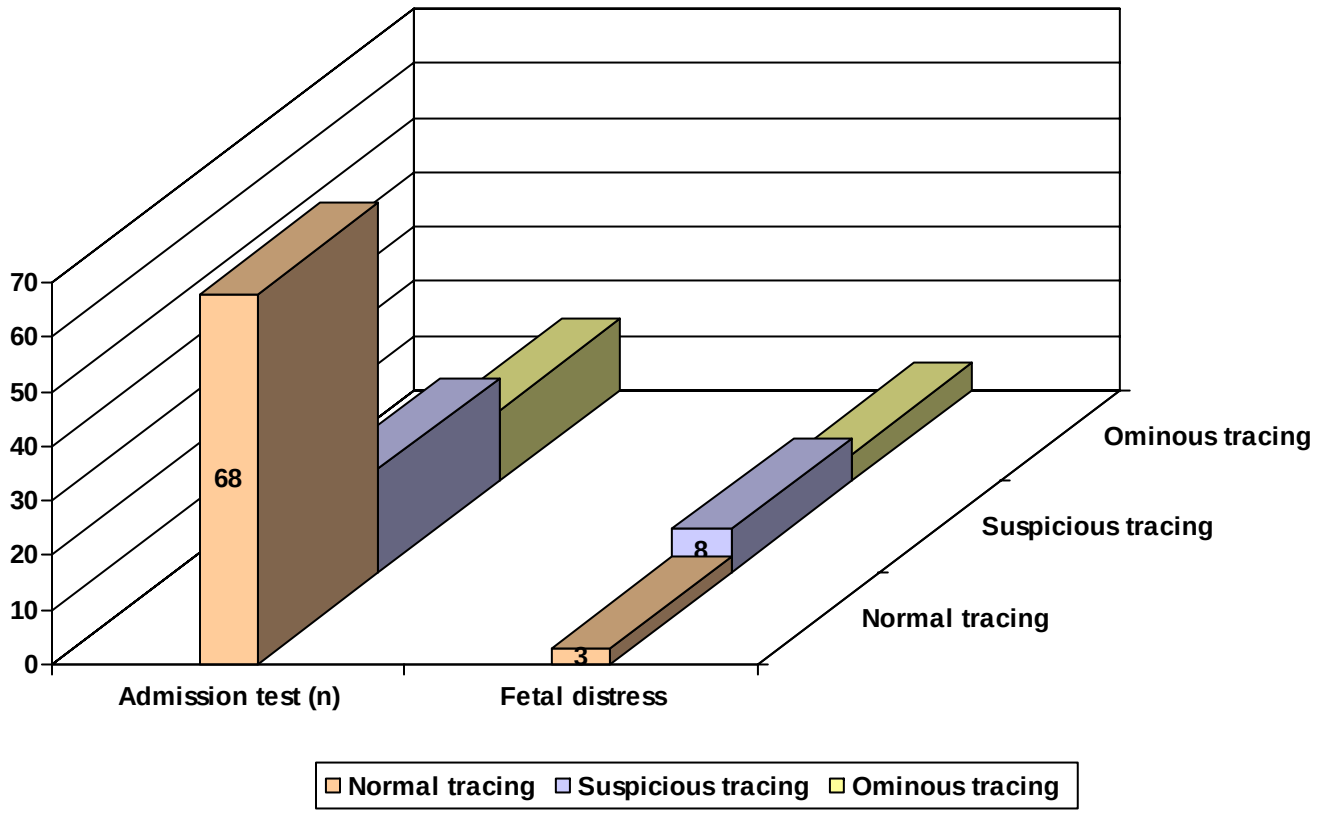
Apgar Score according to CTG pattern in High Risk Cases



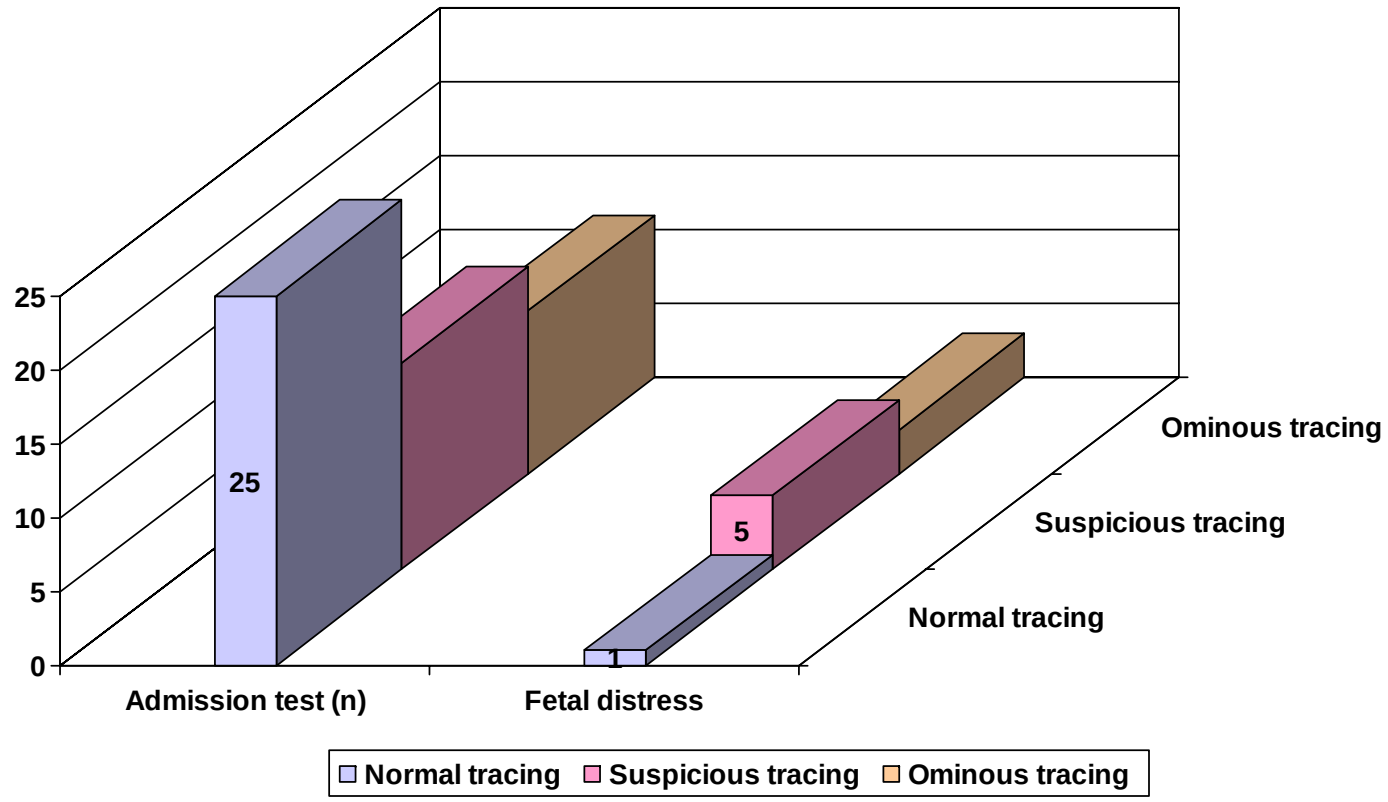
Apgar Score according to CTG pattern in Low Risk Cases



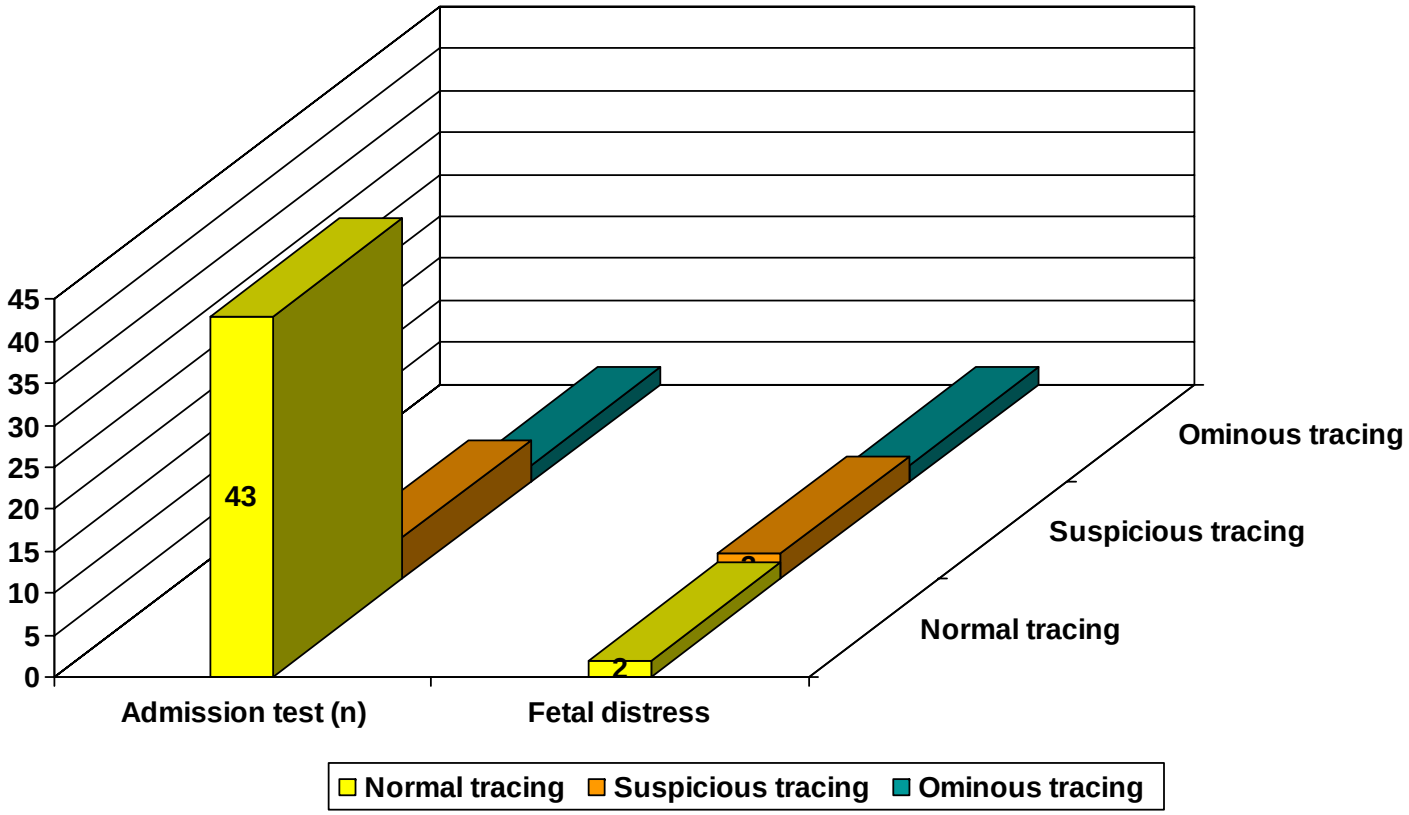
Results of AT in relation to the incidence of fetal distress



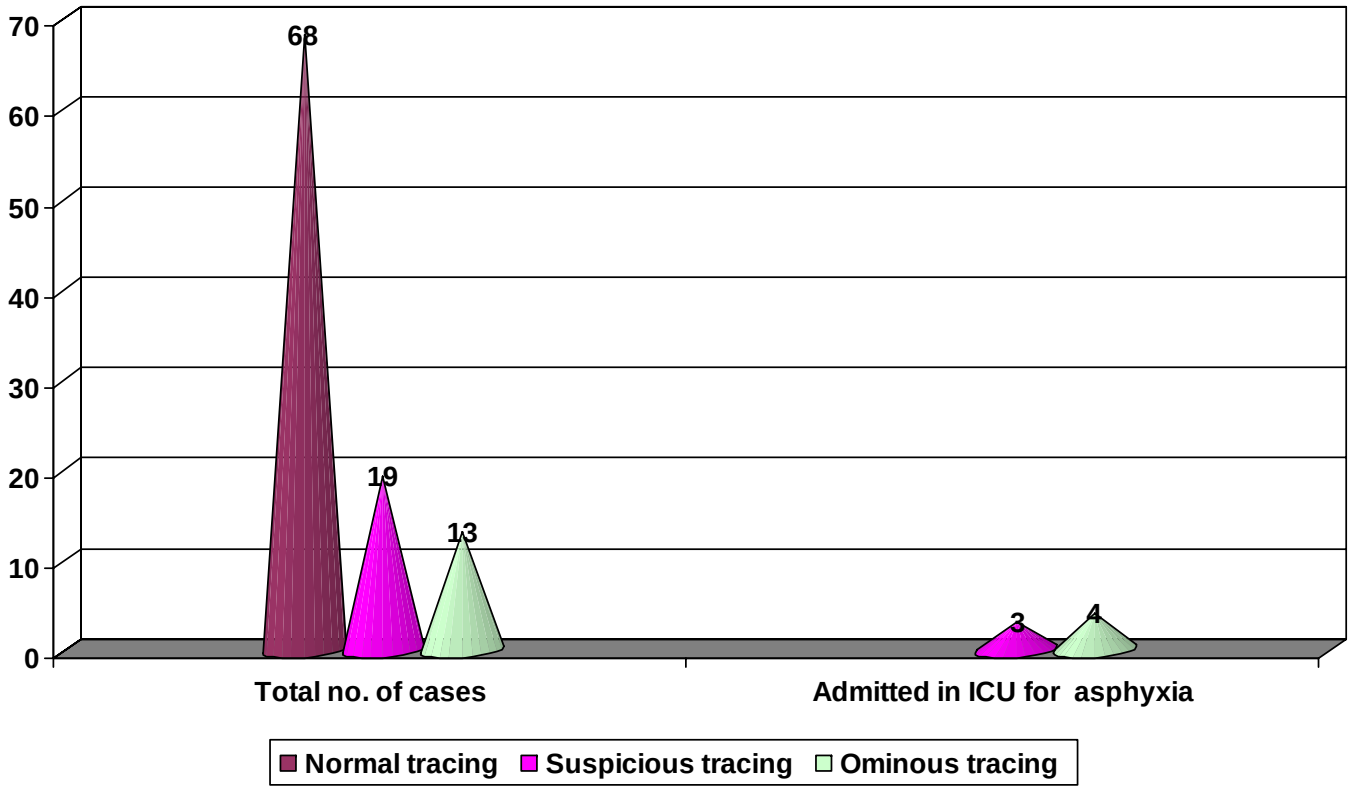
Results of AT in relation to the incidence of fetal distress in high risk group



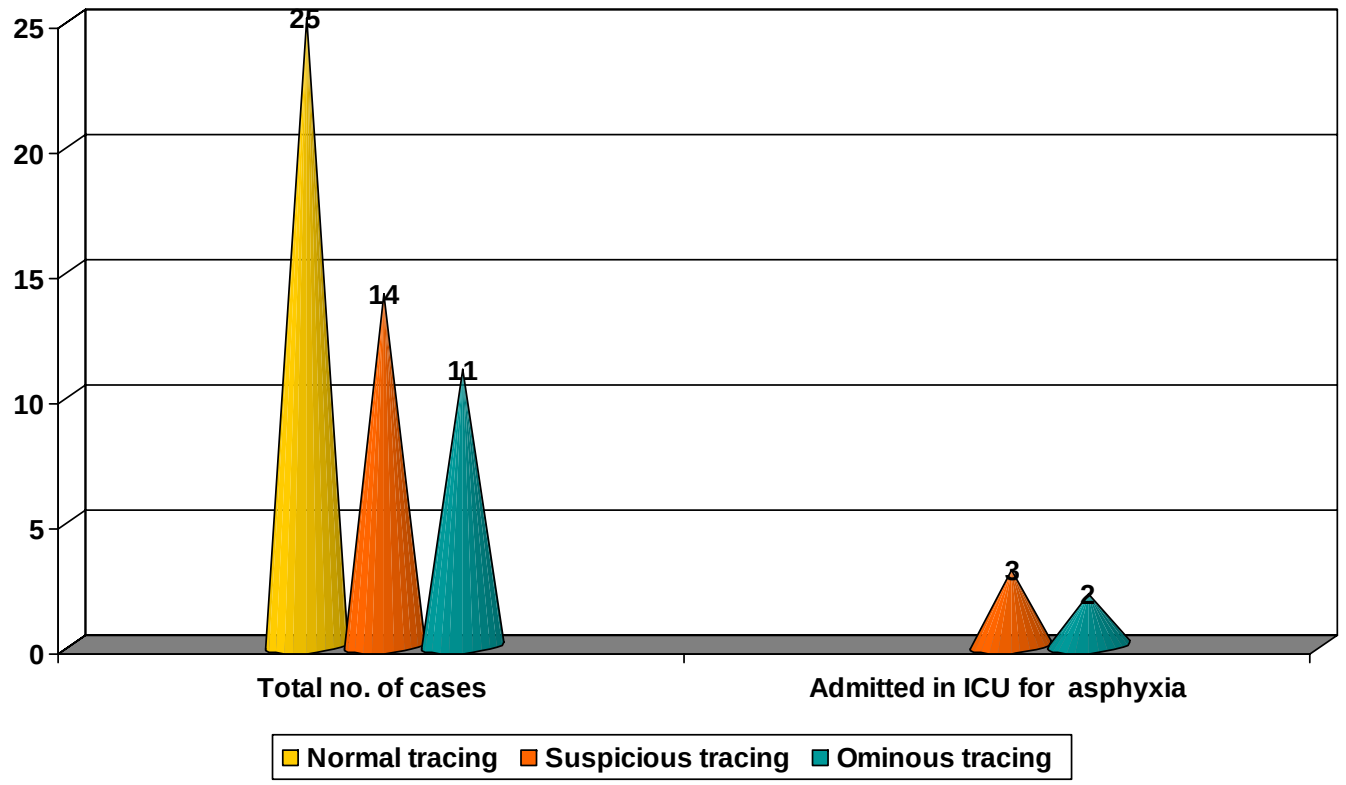
Results of AT in relation to the incidence of fetal distress in low risk group



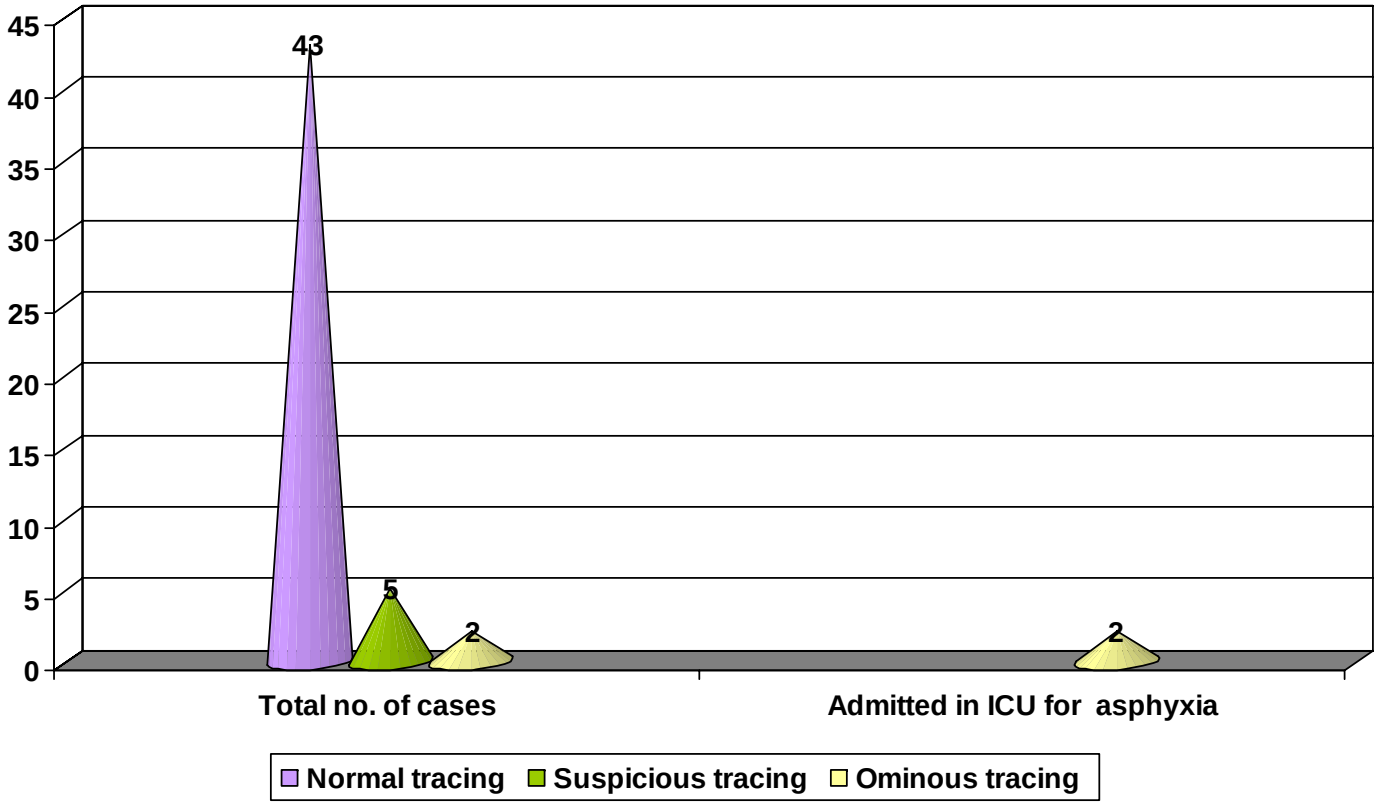
Neonatal ICU admission (Total cases)



Neonatal ICU admission in high risk group



Neonatal ICU admission in low risk group



Efficacy of CTG